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THE INFLUENCE OF FEED CONTROL ON THE GROWTH PATTERN AND PRODUCTION PARAMETERS OF BROILER CHICKENS

DE INVLOED VAN VOEDERSTURING OP HET GROEIPATROON EN DE PRODUCTIEPARAMETERS BIJ VLEESKIPPEN

door

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Chapter 1

INTRODUCTION

Archaeological evidence shows that chickens were firstly domesticated from the red junglefowl *Gallus gallus* in the Southeast of Asia well before 6000 before Christianity (BC). They were taken north to become established in China around 6000 BC. Further spread of chickens eastward from their centre of origin is only poorly known. West and Zhou (1989) speculated that they were introduced to Japan via Korea (during the period 300 BC and 300 after Christianity (AC)). The spread westward is better documented. The domestication in India was established, around 2000 BC. It is however not clear if this was independent or as a diffusion from Southeast Asia.

Several routes across Asia and Europe have been postulated (Figure 1). The Iron Age (1000 BC – 0) seemed to be the main period of dispersion of the domestic chicken (*Gallus domesticus*) throughout Europe. However, they were already present in some of the European areas during the late Neolithic (2500-1800 BC) and early Bronze Age (1800-1000 BC). West and Zhou (1989) concluded from their research that the dispersion to Europe has taken place from China via Russia. Others proposed a route from Iran to the Mediterranean (Figure 1).

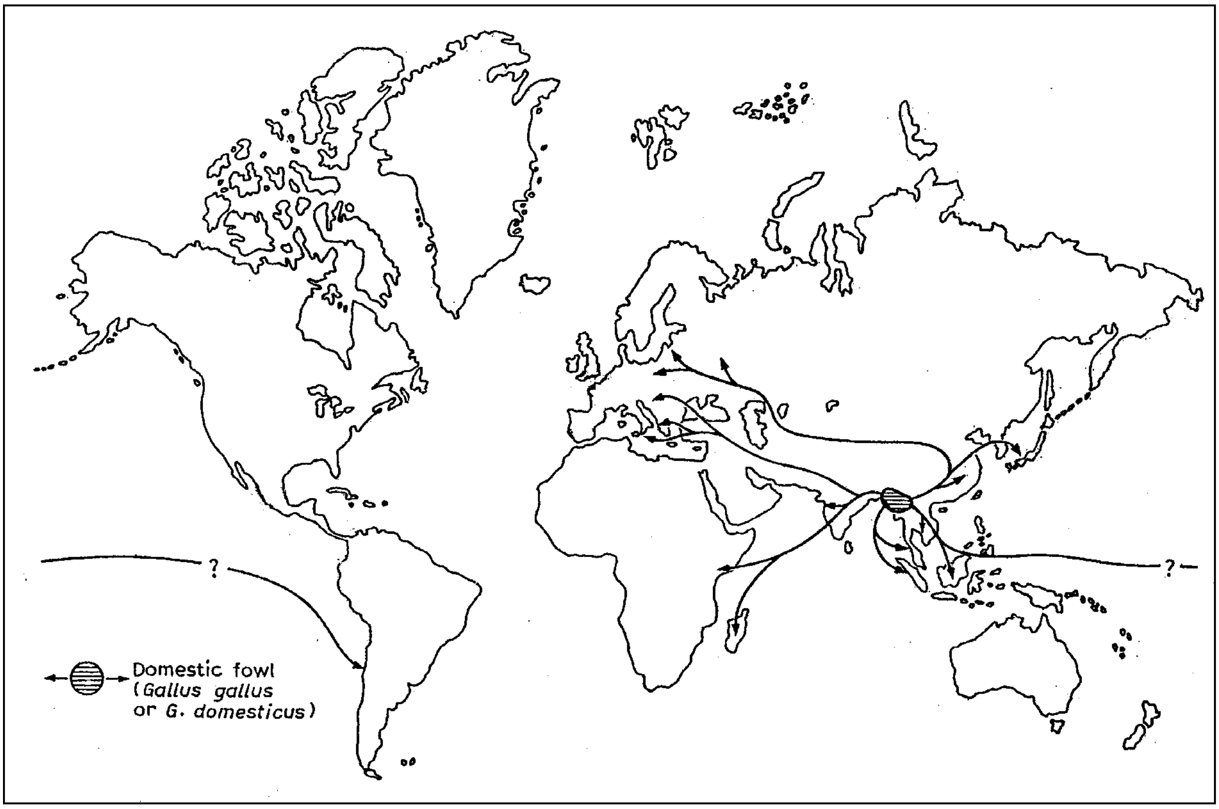

Figure 1 : Postulated early dispersion routes for domestic fowl (Crawford, 1995)

Out of old manuscripts it was clear that the ancient Egyptians, around 1500 BC, knew the chickens. Later they were introduced more in the west via North Africa. It were probably the Greeks who brought them further to Italy. However, before the year 1000, keeping chickens mainly belonged to monasteries and nobility. It was King Charlemagne who introduced the keeping of fowl also to the common people.

In the Middle Ages, domestic fowl was kept in backyards mainly for the production of eggs. Only spent hens and 'out of use'-cocks, and sometimes larger pullets, were used for meat consumption. The holding of chickens especially for egg production was common practice until far into the twentieth century.

In the late 19th century, the existing breeding was focused on perfection of feathers and forms to achieve success at exhibitions, not at all for meat production. Most of this multitude of breeds and varieties only continues today in the hand of fanciers. Only a very few emerged as profitable layers and meat birds.

Over the last 50 years, selection programmes for fast growth and improved feed efficiency have been highly successful in these meat-type birds. Combined with improvements in the feed industry, increasing both nutritional and physical density, growth rate has more than doubled.

These rapid growth rates and heavy body weights, however, are correlated with a significant increase in mortality due to metabolic diseases and an increased incidence of leg problems. Indeed, in its natural habitat, chicken muscle-skeletal development is in proportion to the relative lung size and the heart capacity to meet all physiological requirements under normal growing conditions. On the other hand, the genetic selection programmes have been concentrating on the rapid development of muscle tissues without regard to other body organs. Moreover, the fast growth rate puts high demands on the supply of nutrients and oxygen to the organs. A disagreement between the supplies of the tissues and their demands results in metabolic-related disorders (Decuypere et al., 2000).

In the broilers, 'sudden death syndrome' (SDS) and pulmonary hypertension syndrome resulting in ascites, are the main metabolic disorders. The main leg problems involve tibial dyschondroplasia (TD) and bone deformities and fractures. Unfortunately, these losses can have a main impact on the income of the farmer and are cause of a worse animal welfare.

One approach in controlling these negative selection responses is to restrict growth in the early phase of life. Counting on compensatory growth in the later stage of life, similar final body weights can be reached. According to the results of many researchers (Robinson *et al.*, 1992; Carter *et al.*, 1994; Leterrier *et al.*, 1998), indeed, it is possible to reduce the incidence of skeletal disorders by using these feeding programmes. Many reports also show a reduced mortality due to ascites or sudden death syndrome after reducing initial growth rate (Bowes *et al.*, 1988; Albers *et al.*, 1990; Arce *et al.*, 1992; Fontana *et al.*, 1992; Classen *et al*., 1994; Van Harn and Fabri, 1995; Van Middelkoop, 1997; Gonzales *et al.*, 1998; McGovern, *et al.*, 1999; Acar *et al.*, 1995 and 2001; Urdaneta-Rincon and Leeson, 2002). However, others could not confirm these findings (Proudfoot *et al*.,1983; Mollison *et al*., 1984; Deaton, 1995; Madrigal *et al.*, 1995; Van Harn and Van Middelkoop, 1998). It is clear that literature is rather equivocal on the effects of feed restriction programmes on losses due to metabolic diseases or leg problems. Moreover, also the effect on zootechnical performances and carcase composition are rather variable (see further). More research is needed.

Aim of the study

1. To investigate whether feed restriction programmes described in literature are still an economically feasible management tool for the now available broiler lines and the currently applied management techniques.

   Indeed, as genetic progress is very pronounced, it could be hypothesised that earlier proposed restriction programmes have become inadequate to reduce metabolic diseases. Moreover, due to the increased growth rates, the duration of the production process (from hatch to slaughter age) has been shortened. In this way, the available time to catch-up for restricted birds has shortened extremely. Also in this context, the need exists to re-evaluate the earlier described feeding programmes (Chapters 4 and 5).

   To meet the consumer's demand for a good tasting piece of meat, in all circumstances, precautions should be taken against impaired meat quality. Reports from the poultry industry suggest there is a higher incidence of meat quality problems in modern commercial broilers (Barbut, 1997; Wilkins *et al.*, 2000). On the other hand, the effect of feed restriction programmes on the resulting meat quality is for the most part lacking in literature. Research was carried out to investigate meat quality of feed restricted broilers (Chapters 4 and 5).
2. To investigate the effect of feed restriction programmes on N-retention.

   Indeed, little is known in literature about the N-retention in function of age on the one hand and the effect of feed restriction on the other hand (Chapter 6). With compensatory growth, a better feed efficiency is expected (see further). As protein is one of the most expensive elements in the cost of a complete feed, it is important to use protein in particular as efficient as possible. Moreover, a better protein conversion contributes to the alleviation of environmental N-pollution.

3. To investigate some additional factors in explaining the variable effects in literature of feed restriction programmes.

   According to literature, the described variability in results of an early feed restriction can be explained by a number of factors such as nature, timing, severity and duration of the restriction or genetic factors such as strain and sex. Still it seems that these parameters are not sufficient to explain all of the published variation. The interest was to find some additional factors of influence when describing early feed restriction and compensatory growth. The impact of dietary protein content, one-day old chicken weight (as a possible indication of chicken quality) and feed structure were examined in the present work (Chapters 7, 8 and 9, respectively). As all these parameters are correlated with the growth performances of the birds, it might be postulated that they are also involved in compensatory growth capacity.

In conclusion, the purpose of this work is to provide new information on growth control of modern broiler lines which can be used as a guide for the farmer to grow his broilers as optimal as possible. In this way, this thesis may contribute to improved economic returns for the farmer with an increased respect for animal welfare. Together with the alleviation of environmental pollution (due to the increased N-retention), this research on feed control programmes may contribute to the development of a more sustainable agricultural production.

Chapter 2

LITERATURE STUDY

1. Poultry production in general

The poultry meat husbandry has become one of the most dynamic areas of animal production. Indeed, poultry meat is universally accepted, there are no religious or cultural barriers to poultry meat consumption. Poultry meat is considered as a healthy, nutritious and affordable choice among the available meat sources. Moreover, poultry meat is easy to prepare and exists in a wide variety of end products.

Total world production reached 71.6 million tonnes in 2002, which is more than eight times the level reached in 1961. According to FAO forecasts, the output will continue to rise in the near future. Moreover, almost 86 % of all poultry meat is chicken meat, a percentage that has been rather constant during the last 40 years. The USA, the European Union and China are, and will probably continue to be, the world’s leading producers, accounting for more than half (56 %) of the world total (Gillin, 2002).

The Western European countries contributed 27 % of the output of developed countries in 2001 (Gillin, 2002). Table 1 shows that the United Kingdom, France and Spain are the leading countries followed by Italy, the Netherlands, Germany and Belgium. From these data it becomes clear that the ranking of the production volume does not necessarily reflect the ranking according to population. Per capita consumption and the rate of self-sufficiency are of course also important steering factors.

Tabel 1 : Broiler meat production in the EU in 2001 (ZMP-Bilanz, 2002)

Country	Broiler meat (ton)	% of EU
United Kingdom	1,211,000	19.1
France	1,104,000	17.4
Spain	957,000	15.1
Italy	710,000	11.2
The Netherlands	620,000	9.8
Germany	535,000	8.5
Belgium/Lux.	285,000	4.5
Portugal	229,000	3.6
Denmark	188,000	3.0
Greece	159,000	2.5
Ireland	94,000	1.5
Sweden	92,000	1.5
Austria	80,000	1.3
Finland	63,000	1.0
Total	**6,327,000**	**100**

An overview of the poultry meat consumption in function of time for some European countries is given in Figure 2. Mean consumption in Europe in 2000 was 16 kg/person/year. In 2002 it dropped by 2.3 % compared with the previous year but increased by 4 % against 2000 (Feedinfo News Service, 2003). Also in Belgium, poultry production has become a very important branch in animal production.

An evolution of the Belgian broiler sector is given in Figure 3 for the period 1991 to 2001.

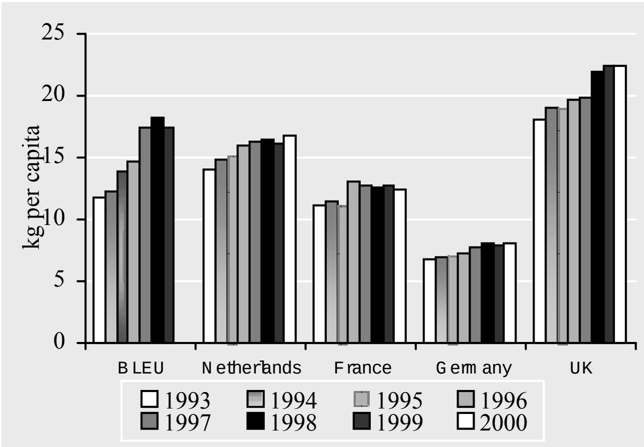

Figure 2 : Evolution of the poultry meat consumption in function of time in some European countries (Source : ZMP-Bilanz)

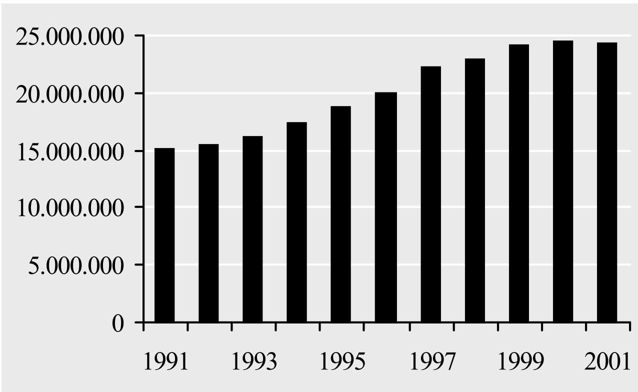

Figure 3 : Evolution of the number of broiler chicken places in Belgium in function of time (15 may count) (Source : N.I.S.)

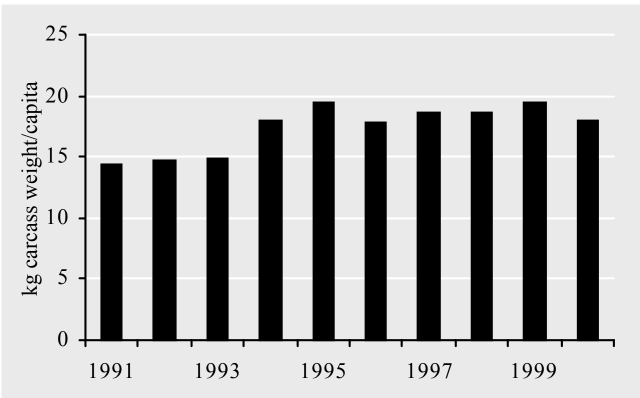

Figure 4 : Evolution of the Belgian poultry meat consumption in function of time (kg carcase/capita) (Source : C.L.E.)

Livestock in total takes about 62 % of the production value of the Belgian agriculture (2000).

This 62 % is divided in 23 % for beef+milk, 30 % is taken in by pig production. Poultry production counts for 8 % (CLE, 2001). The turnover of the poultry meat production in 1999 reached 206 million euro (CLE, 2001). The self-sufficiency in Belgium for poultry meat is 157 % (2000).

The evolution of the apparent meat consumption (flesh, bones and offal) in Belgium in function of time is given in Figure 4. In 2000, yearly poultry meat consumption in Belgium was 18 kg/per person. Although absolute figures know a descending trend, the relative share in the total meat consumption increases with time. This can be explained by the decreasing consumption of beef meat since 1996 and a decreasing consumption of pork meat since 1993. These trends evolve at a faster rate than poultry meat. It can be concluded that poultry meat production is a very important branch of animal production.

2. Growth of the broiler chicken

Growth is a very complex process. It is dependent, next to the genetic constitution of an individual, on level of nutrition and numerous environmental factors. Growth of a whole animal, its organs or tissues, is regulated by a coordinated, integrated control system. As growth is physiologically seen a very wide concept, it can be described in different ways.

2.1. Growth curves

When studying the influence of feed control on the growth pattern of broiler chickens, it is important to be able do describe the growth of birds very accurately. Body weight measured at a specific age is probably the most frequently used method of describing growth, as it is relatively easy to measure as opposed to other measures of growth. The data obtained by weighing the birds can be used in a mathematical model, describing the growth of the birds with advancing age.
A growth function is an analytical function written as the following equation : $W = f(t)$, where W is the weight of the chicken at time t. In other words, a large amount of data can be represented by simple functions and thus become easily manageable information.
The following linear equation could be used :

$W = a.t + W_0$ (W_0= weight at hatching)

In this case, it is assumed that equal increments in live-weight occur in equal increments of time. The slope ‘a‘ can be described as the rate of live-weight gain. It is clear that this linear model is an oversimplification of the reality. In general the growth curve of a broiler is, however, sigmoid. It is characterised by an accelerating growth phase from hatching, a point of inflection at which the growth rate is at his maximum followed by a phase of decelerating growth. Moreover, the curve tends to an asymptote being the mature weight of the bird.
Since the 19th century, many researchers have been developing mathematical models to describe the growth of birds. Well known models are :

Richards : $W = A\,(1 \pm e^{-Kt})^{M}$

Gompertz : $W = W_0\, e^{(L/K)(1-e^{-Kt})}$

Brody : $W = W_0\, e^{(Ct)}$ $(0 \le t \le t')$

$W = A\,(1 - B\,e^{-Kt})$ $(t' \leq t)$

Von Bertalanffy : $W = A\,(1 - B\,e^{-Kt})^3$

Logistic : $W = A\,(1 + e^{-Kt})^{-1}$

with W = liveweight, t = age, W_0 = weight at hatching

with parameters to be estimated: A (asymptotic adult weight), B, C, K, L, M, t' (age of puberty) (Fitzhugh, 1972; Wilson, 1977; Tzeng and Becker, 1981; Gille, 1998).

In the study of Tzeng and Becker (1981) different models were compared for their fit to weight data. The equation with the lowest F-value was assumed to provide the best fit of the data. Out of this research it followed that the Gompertz equation had the best fit. Also in the study of Knízetová *et al.* (1991), Hruby *et al.* (1996) and Hurwitz and Talpaz (1997), the Gompertz function was preferred for describing the growth of poultry with a minimum of parameters up to the usual slaughtering age.

These empirical models are rather restricted in use, since they consider the bird as a system with an output only. In reality, birds are subjected to a lot of factors (feed, temperature, lighting programmes and other management factors) of which the influence can not be predicted with such a model.

First, this view of animal growth as an input-output system has led to the development of growth equations which relate increase in mass to feed intake (Spillman and Lang, 1924; Titus *et al.*, 1934 : quoted by Wilson, 1977). However, as the quality of the feed has a major influence on growth, this factor was incorporated in the more recent models (Parks, 1973: quoted by Wilson, 1977). The next advance was established with the model of Whittemore and Fawcett (1976) which predicted the growth of the pig as a response to its known diet in terms of both the composition of the weight accretion and the rate of (controlled) feeding and this for different environmental temperatures. An overview of the evolution of mathematical models in broiler raising is given in Zoons *et al.* (1991). Broiler growth simulation is possible using the EFG model (created by Gous, Emmans and Fisher) considering many other influences on growth than nutritional ones (Nicholson, 1996). However, when using physiological knowledge to develop models, they may be indeed more accurate and suitable for predictions but are usually also very complicated. However, in the course of this work, only the description of the difference in growth between *ad libitum* fed and restricted birds is necessary, so the Gompertz equation (W versus time) seemed very suitable and easy.

2.2. Allometric growth

Analysis of growth curves as single entities (one curve for the whole animal) is not the only method of growth analysis. Indeed, since a long time, it has been recognised that growth is not only an increase of weight in function of age, but that also a difference in conformation takes place. For this, the study of proportional or allometric growth was introduced. Huxley's allometric growth equation, $Y = a X^b$ or $\ln Y = \ln a + b \ln X$, has been used frequently to describe the relation between the weight of a part of the body (Y) against the weight of a bigger part X (e.g. the body weight) during a well defined growth trajectory. The coefficient b is defined as the allometric coefficient. If b > than 1, then the weight of part Y is growing at a faster rate than the remainder of the body, and vice versa. If $b = 1$ then the weight of part Y remains constant as the body grows. Vital organs such as heart, liver, digestive organs are early maturing ($b < 1$). Lungs know a rather isometric development ($b = 1$) while breast meat and abdominal fat pad are late maturing ($b > 1$).

2.3. Endocrine regulation

There are a variety of hormones which are involved in the regulation of the growth process. Moreover, growth control by hormones is not only dependent on interactions between the hormones themselves but is dependent on the presence of receptors. The ability of a hormone to influence tissue metabolism and growth depends on the circulating levels of the hormone, its rate of delivery to the target tissue, the number and affinity of hormone receptors present and the responsiveness of postreceptor events to hormone action.

Somatotropic hormone (STH), often called growth hormone, is, as his name may let expect, an extremely important hormone with a multiplicity of effects. However, it cannot be considered alone as being of primary importance because its growth-promoting role is not solely of direct action but also as a mediator of other factors that act at tissue level. In turn, other factors mediate in its primary growth-promoting role.

Birds have two insulinlike growth factors (IGF-I and IGF-II) that are important regulators of cellular differentiation, proliferation and growth of tissues. The liver is the main source of production but there is also a significant local release by many different tissues. It seems highly likely that both the systemic and local releases have a role in mediating the action of growth hormone.

Also thyroid hormones play an important role in growth and development. The predominant iodothyronine secreted by the thyroid glands is thyroxine (T_4), a pro-hormone which is converted to the active form triiodothyronine (T_3). They stimulate oxidative metabolism and anabolic functions of cells by regulating oxygen consumption, mineral balance and the synthesis and metabolism of protein, carbohydrates and lipids.

Also insulin (pancreatic hormone), glucocorticoids (e.g. corticosterone and cortisol), gonadal steroids (androgens, oestrogens) are involved in growth control with a direct or indirect effect. For a more detailed description of the hormonal regulation of growth the reader is referred to Lawrence and Fowler (1997) and Cogburn *et al*. (2000).

2.4. Genetic selection

Genetic selection as a means of improving the growth rate of broilers has been highly successful over the past 50 years. Indeed, meat-type chickens have been selected for rapid growth more intensively than any other species. From the late forties of the past century on, genetic selection diverged between layers and meat type broilers. This was the start of a tremendous evolution in the poultry husbandry. Next to selection, there was a major evolution in the poultry nutrition. Research was focussed on the determination of the nutrient requirements for production and maintenance. The following figures illustrate this evolution : in the forties it took the farmers more than 100 days to rear a broiler of 2 kg, nowadays it takes them only 37 days!

The annual rate of genetic progress is mentioned in Table 2. Growth curves of Ross broilers as available in 1980, 1990 and 2000, are shown in Figure 5. It can be concluded from these figures that each 10 year interval has taken about 6-10 days of the time to reach 2 kg, as well as giving a bird with a higher mature body size.

Table 2 : Current annual rate of progress in genetic selection (after McKay and Keaveney, 1998)

Annual rate of genetic progress	
Live weight (42 d)	+ 55-60 grams
Feed conversion to 2 kg	- 0.04-0.05
Eviscerated yield at 2 kg	+ 0.20-0.25 %
Breast meat yield at 2 kg	+ 0.25-0.30 %

These advances are amazing, however it is clear that limits will be reached. At present there is no indication of a reduction in genetic variability for the main broiler traits (Pollock, 1999), but the ongoing pace of progress can undoubtedly not be maintained until the very end.

This could be illustrated when extrapolating the current progress in future. Keeping up the same progress in the future would mean that within 40-50 years, a body weight of 2 kg will be reached …..at hatching! At the present time, however, some biological limits seemed to be

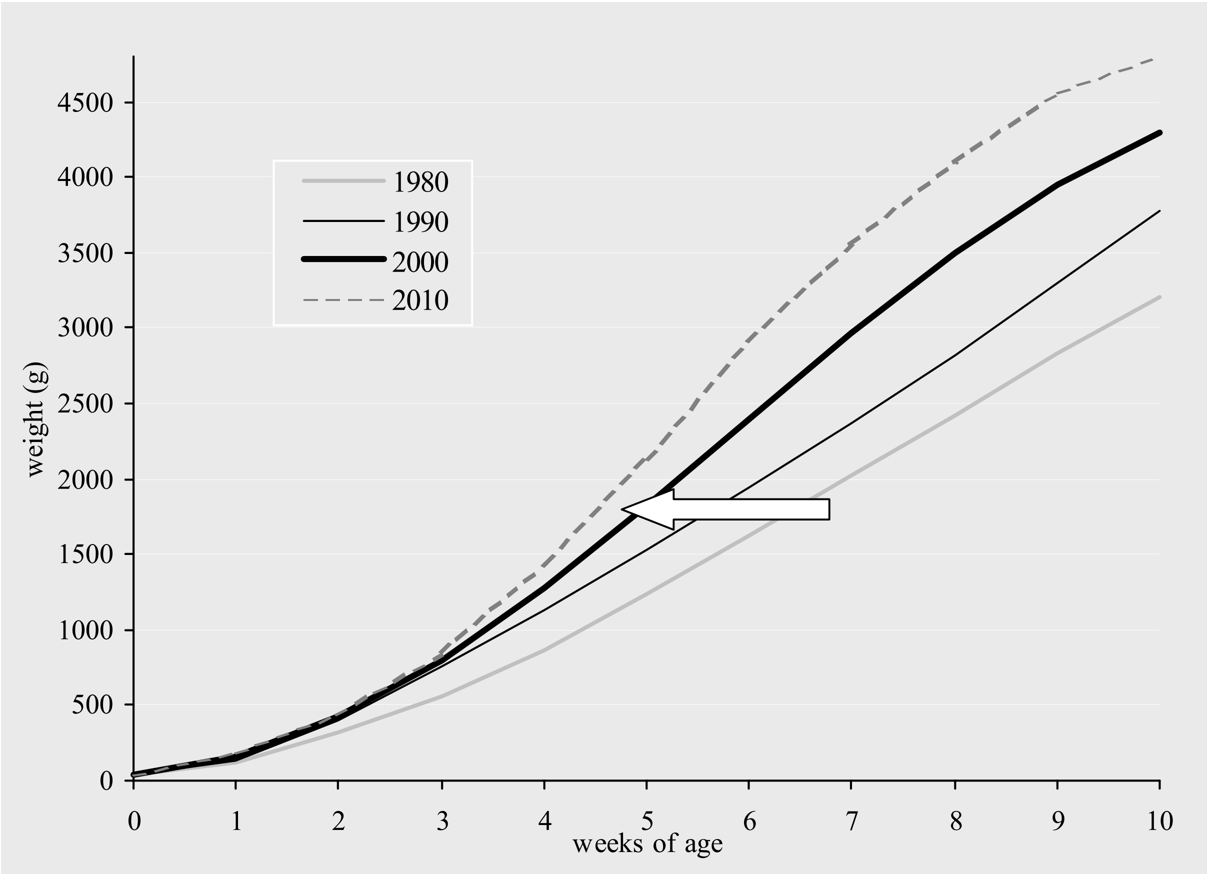

Figure 5 : Growth rates in function of age of Ross broilers for the years 1980, 1990, 2000 (based on data of Ross Breeders Ltd.) and as expected in the year 2010 (extrapolation). Indication of the age reaching 2 kg of body weight.

reached already. Indeed, modern broiler lines are characterised by an increased incidence of leg problems and metabolic disorders. A comparison between meat- and egg-type chickens reveals that the selection for increased body growth was not accompanied by an appropriate development of the so-called ‘supply’ organs (Figure 6; after Plavnik and Hurwitz, 1983). Indeed, growth rate of the meat-type breed was about three times higher in comparison with the egg-type breed. On the other hand, the relative weight of many organs associated with metabolism, ‘supply’-organs (spleen, pancreas, liver, gizzard, heart), showed no corresponding difference to accommodate this difference in growth (Figure 6). Although interactions with dietary and environmental circumstances can not entirely be excluded, the differences in proportion of the different organs are mainly related to the inherited potential of each breed. Also when comparing two meat-lines, Boa-Amponsem *et al.* (1991) described similar findings. When comparing a 1957 Athens Randombred Control strain, with a 3.4

times lower body weight, a higher percentage of heart and lungs was found when comparing with the 1991 Arbor Acres commercial broiler (Havenstein *et al.*, 1994). These findings clearly show that an imbalance between 'supply'- and 'demand'-organs (muscles) has been created, which may explain the increased rate of metabolic disorders in the modern poultry strains.

3. Metabolic disorders

The aetiology of sudden death syndrome (SDS) and ascites in broilers are closely related (Squires and Summers, 1993). Moreover, the causes seem multifactorial as diet, environmental and genetic factors, but also their interactions play an important role (Decuypere *et al.*, 2000). They both involve cardiovascular problems.

3.1. Sudden death syndrome

Sudden death syndrome is next to ascites, one of the main disorders. It is indeed typical for fast growing broiler chickens, especially males. The normal incidence is 1.5-2.5 % of the flock (Leeson *et al.*, 1995). It is typical for the period from d 21-28 although it may occur as early as 3 days of age and continue throughout the entire growing period. Birds appear to be in a healthy state prior to death. They are usually well fleshed and have a weight above the flock average. Death occurs within a few minutes and the birds are usually found on their backs (current name : flip-overs).

At dissection, no specific changes in the tissues or blood profile are found. Feed is present along the entire digestive tract. Research did not show any clear correlation between a dietary nutrient and/or environmental factors and the onset or incidence of SDS. The incidence of flip-over, however, is uncommon where low density feeds or native birds are used. In other words, it is most likely that SDS is related to a fast growth rate, and as such, management techniques to reduce the juvenile growth most likely will offer the best preventive scenario (Bowes *et al.*, 1988).

3.2. Ascites

The ***'ascites syndrome'*** also, is most prevalent in fast growing male broilers, especially if they are maintained at high altitude or a cold environment (due to a O_2-shortage). However, ascites

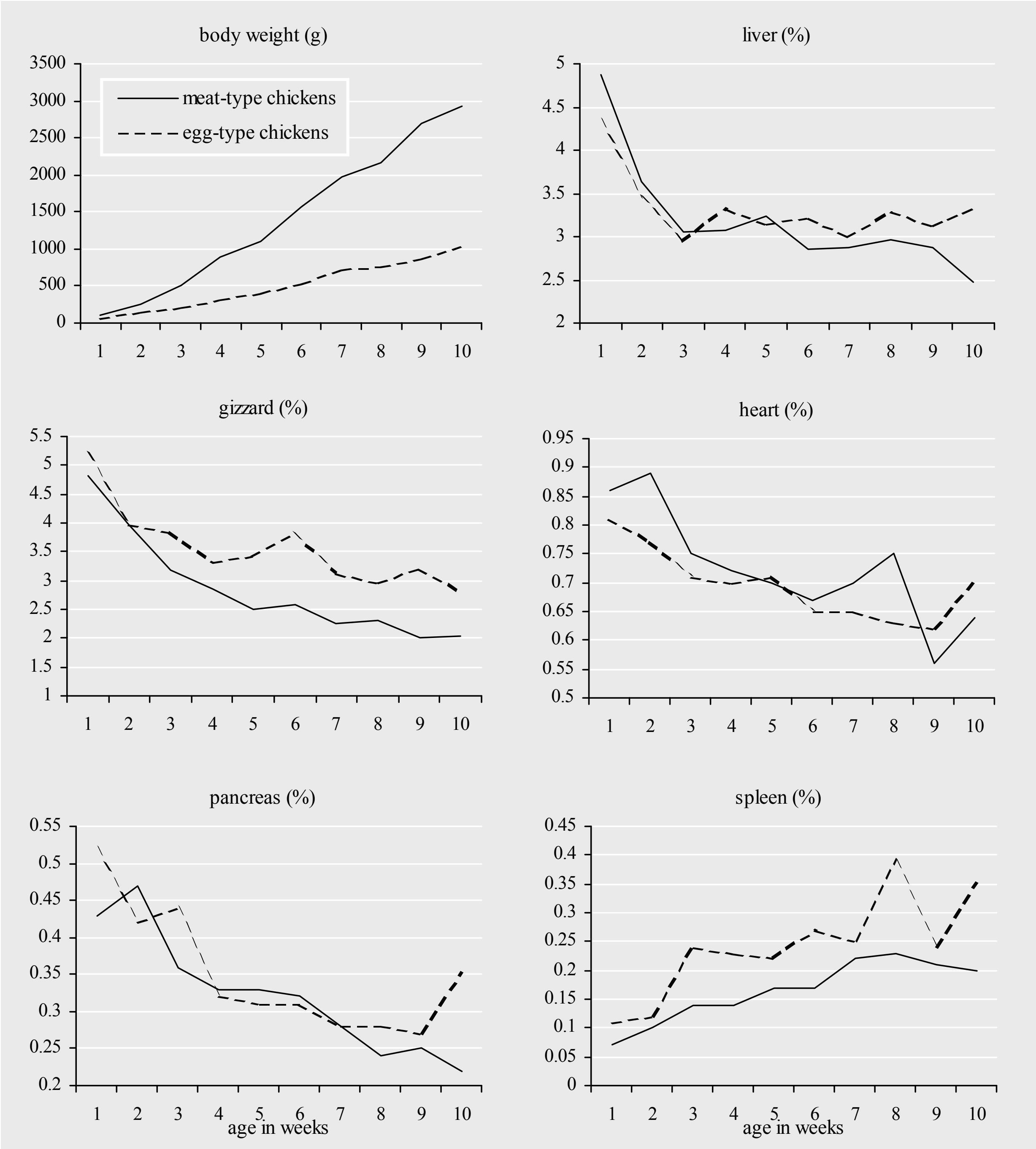

Figure 6 : Comparison between meat- and egg-type chickens of body weight and relative proportions of liver, gizzard, heart, pancreas and spleen as percentage of body weight (after Plavnik and Hurwitz, 1982)

also seems to cause an increased death rate in common poultry houses. A study of Maxwell and Robertson (1997) revealed that the incidence of ascites in the UK in 1993 was 1.4 %. Ascites is characterised by accumulation of fluid in the abdominal cavity leading to death or carcase condemnation. Symptoms and death occur mainly from the age of 3 weeks on although the predisposition for the development of the syndrome already occurs in the first weeks of life. There is no treatment or intervention that can reduce its incidence in the later stages of life. Fundamentally, ascites results from the inability of the modern broiler to provide tissues with an adequate supply of oxygen (Decuypere et al, 2000). As mentioned before, due to the genetic selection, the relative conformation of the broiler has changed with e.g. a major increase in percentage breast meat (Figure 7). Relative to body weight, these birds have a much lower lung volume (estimated at 25 % less) than the original junglefowl. Moreover, the lungs of birds are firm and fixed in the thoracic cavity. They do not expand and contract with each breath as mammalian lungs do. In other words, the rapid growth rate, high muscle yield and high metabolic rate, do not only require a higher oxygen supply, it has to be provided by a smaller long volume.

In order to meet the demands, the bird attempts to pump more blood through the lungs, which places extra stress on the right ventricle of the heart. An increase in blood viscosity further contributes to right ventricle hypertrophy. Indeed, anoxia in birds stimulates the kidneys to produce erythropoietin which, in turn, stimulates the production of red blood cells (essential for the transport of oxygen) in the bone marrow. This results in higher haematocrit values, which are accompanied by an increase in the viscosity of the blood. The resulting right ventricular hypertrophy leads to a failure in the closure of the right valve between the ventricle and the atrium. As a consequence, a volume of blood re-enters the atrium with each heartbeat. This results in a substantial increase in the venous pressure in the portal and hepatic veins which forces plasma fluid (oedema) out of the vessels, into the peritoneal spaces. This condition is called ascites (other name : water belly).

3.3. Skeletal disorders

Although many of the components causing leg disorders can be attributed to malnutrition, leg weakness can still be an important economic and welfare problem even when supplying all necessary nutrients. Next to nutrition, also infectious diseases or toxins can induce leg problems. This work, however, will focus on the metabolic disorders. Due to high growth rates, there is a considerable strain on leg muscles and bones during the last half of the growing period (Lilburn, 1994). There seems to be two general classes of ‘metabolic’ leg problems reported in the literature. The first is tibial dyschondroplasia (TD).

Tibial dyschondroplasia is characterised by an abnormal cartilage mass in the proximal head of the tibiotarsus (Figure 8). It occurs as a result of failure of proliferating chondrocytes in the growth plate to hypertrophy to allow vascular penetration and the normal production of bone (Julian, 1998). Mild and moderate lesions may not cause lameness, although the proximal end of the tibia may be enlarged. Severe lesions, however, cause weakening of the proximal tibia

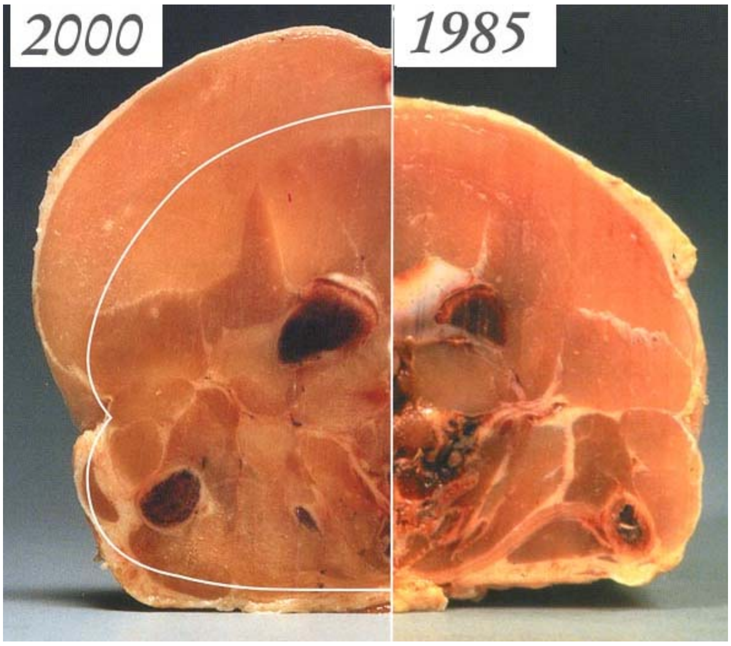

Figure 7 : Illustration of the effect of 15 years of genetic selection on breast meat percentage in broiler chickens (Decuypere, 2003)

which is compressed by body weight as the bird walks, causing painful lameness (Julian, 1998). The weakened proximal tibia may be pulled backward by the strong gastronemius muscle, causing deformity, or the large cartilage mass may develop in avascular necrosis eventually followed by a spontaneous fracture of the proximal tibia. TD is most specifically related with rapid growth. Indeed, it is common for the rapid growing modern broiler

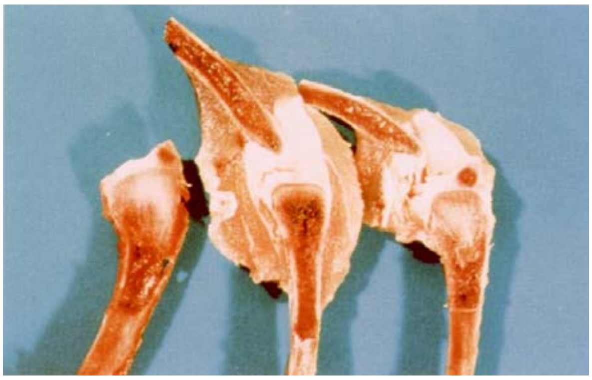

Figure 8 : Example of tibial dyschondroplasia (middle is normal)

Figure 9 : Leg abnormalities

but very rare in other birds. In general, it is next to genetic factors, also linked with dietary electrolyte imbalances (high chloride concentrations) and low Ca/P-ratios. Nevertheless, reducing growth rate seems to have a preventive action (Robinson *et al.*, 1992).

A second general category of leg weakness consists of ***angular and torsional deformities of the tibiotarsal and the metatarsal bones and bone fractures*** (Figure 9). De major angulation occurs at the distal end of the tibiotarsus but lesser angulation may also occur in the proximal tarsometatarsus. Although angulation is the major deformity, some rotation of the distal tibia may also occur (Riddell, 1992). The pathogenesis and aetiology of these deformations is however yet poorly defined.

4. Early feed restriction

4.1. Effects of early feed restriction on the incidence of metabolic disorders

The challenge in growing broilers efficiently involves maintaining a high body weight at market age, avoiding lowered feed efficiency (by excessive fat deposition), lower disease resistance and a higher mortality rate.

Early studies of feed restriction programmes in broilers have been primarily concerned with the lowering of body fat and improving feed efficiency (Griffiths *et al.*, 1977; Moran, 1979; Fisher, 1984) (see also further). Later, also the potentials of these feed restrictions to correct for metabolic problems or skeletal disorders have been reported.

During the intensive growth established in the early postnatal phase (Ricklefs, 1985), also the development of the cardio-vascular system, lungs, the gastro-intestinal tract and the skeleton takes place (early maturing organs, cf. allometric growth). Moreover, the predisposition for metabolic disorders already occurs at this very early age when the metabolic demand is very high (Buys *et al.*, 1998). Early feed restriction, on the other hand, reduces initial growth and, thus, the oxygen requirements of the chickens, which alleviates this metabolic load. In a study of Govaerts *et al.* (2000) it was found that feed restriction causes a shift in nutrient and energy supply giving priority to early maturing supply organs which are more important in the early development. All this illustrates that early feed restriction might re-establish the imbalance between ‘demand’ and ‘supply’ and, thus, prevent metabolic diseases.

A study from Zubair and Leeson (1994b) showed relative heavier digestive organs in broilers restricted to 50 % of voluntary feed intake (6 to 12 d of age) in comparison with control birds. Rosebrough *et al.* (1986) restricted male broilers during the same period and reported relative heavier liver weights on days 14 and 16. McCartney and Brown (1977), Pinchasov *et al.* (1985), Palo *et al.* (1995) and Katanbaf *et al.* (1988, 1989) (females from a broiler-breeder parent stock) also confirmed these findings. These higher proportional weights of the digestive organs of restricted birds further illustrates the theory of repartitioning nutrients in favour of the supply organs (Govaerts *et al.*, 2000). On the other hand, Ballay *et al.* (1992) found little effect of early feed restrictions (different periods between 0 and 18 days of age) on organ weights relative to body weight. Nevertheless, these authors found a less severe response to an *E. coli* inoculation and lower overall mortality in restricted fed birds. Robinson *et al.* (1992) could not establish a significant reduction in mortality due to metabolic diseases although a significant reduction in the incidence of birds culled for skeletal problems was reported when feed-restricted. In the Tables 3-7 an overview is given of the main literature on early feed restriction in poultry. The effect on losses due to metabolic diseases or skeletal problems is indicated when studied. Many reports show a reduced mortality due to ascites or sudden death syndrome. However there are some authors who were not able to indicate a (significant) positive effect. This might have to do with the low number of birds in trial which might have obscured a positive effect of the feed restrictions.

In a study of Scheideler and Baughman (1993), there was a significant positive effect of the feed restriction on the tibia bone ash content. This can indeed be an indication that reducing growth rate in the early stage of life allows a more complete skeletal development before the bird starts gaining meat tissue. Carter *et al.* (1994) and Robinson *et al.* (1992) also found a reduced incidence of leg disorders. In the study of Leterrier *et al.* (1998), an increase in ash-content of the bones could however not be confirmed. None of the parameters describing the morphology, composition and histomorphometry of the tibiotarsi were different between *ad libitum* fed broilers and their restricted counter-parts when compared at equal body weight (Leterrier *et al.*, 1998). These authors had to conclude that the reduced occurrence of varus-valgus deformities (inward and outward angulation of the legs) in slow-growing birds couldn't be related to an improvement in the structure and the composition of their bone tissues.

4.2. Methods to reduce initial growth

When considering feed restriction to reduce initial growth, methods can be subdivided in quantitative and qualitative restrictions. A quantitative feed restriction means that a limited amount of a well balanced diet, with normal nutrient density, is offered to the birds. Qualitative restrictions include diet dilution, chemical methods, deficiencies in certain nutrients or low energy and/or low protein diets.

4.2.1. Quantitative feed restrictions

A simple physical restriction provides a calculated quantity of feed per bird and is one of the most commonly used methods. Usually a certain percentage of the *ad libitum* feed consumption is used. This method has the disadvantage of the frequent weighing of feed. In addition, feeder space must be adequate and the limited amount of feed must be provided evenly and quickly to avoid uneven body weight distribution within a flock. However, nowadays, sophisticated feeding systems with an automatic weigher are available in modern broiler houses, which makes the job of restricting feed quantity no longer a problem. Computer systems are even able to provide the limited quantities, if desired, in several meals a day.

In the past, some trials were carried out with a 'skip a day'-restriction. During the period of restriction, the birds are fed on alternate days. However, in the framework of animal welfare, these kinds of restrictions are not preferable and will not be further discussed in detail in the present work.

4.2.2. Qualitative feed restrictions

Especially when automatic systems are not available, also qualitative feed restrictions can be of use. Diet dilution is a very simple way of lowering energy and protein content of the standard meal. However, to a certain degree, birds are able to adjust their feed intake trying to achieve their requirements. According to the results of Leeson *et al.* (1991), this compensation can be up to 150 % of the normal intake in extreme situations. Thereby, feed structure has a major influence on their capacity to adjust their feed intake. Indeed, the capacity to increase feed intake becomes higher when feeds are crumbled or pelletised (Newcombe and Summers, 1985). In general, when utilising the dilution method to restrict nutrient intake, the inert filler should have enough bulk to limit the physical capacity of the gastro-intestinal tract of the bird.

Fancher and Jensen (1988) first suggested restriction of feed intake by chemical means as an alternative for diet dilution. These authors examined the possibility of using glycolic acid (GA) to restrict voluntary feed intake. According to Pinchasov and Jensen (1989), the inhibitory mechanism of GA acts through the brain serotonergic system. This method of restriction has the advantage of ensuring an even distribution of the diet and appropriate intake of all micro-ingredients such as vitamins, trace elements and anticoccidials.

A sodium-deficient diet also is known to reduce feed intake (Plavnik and Hurwitz, 1990; Meluzzi *et al.,* 1995). An alternative for sodium-deficiency is the use of low protein or low energy diets. When dietary protein is marginally deficient, broilers are able to adjust their feed intake to make up for the deficiency (Fisher, 1984). On the contrary, a more severe dietary protein deficiency results in a lower feed intake (Plavnik and Hurwitz, 1990).

4.2.3. Non-nutritional methods

To complete the picture there should be mentioned that also lighting programmes or eventually low temperature schemes can be used to change the growth curve to a more concave one (Renden *et al.*, 1992; Renden *et al.*, 1993; Buyse, 1991; Buyse *et al.*, 1994a; Buyse *et al.*, 1996; Taranu *et al.*, 1996; Zoons, 1997).

In similarity with the feed restriction methods, alternative lighting schedules can induce a restriction in feed intake and growth during the early age. After an adaptation period, birds are able to adjust their feed intake and realise compensatory growth (Beane *et al.*, 1979; Renden *et al.*, 1992; Renden *et al.*, 1993; Van Harn and Van Middelkoop, 1997; Zoons, 1997). Even heavier weights can be realised using a 1-hour light/1 hour dark treatment in comparison with a continuous lighting schedule (Beane *et al.*, 1979). In general, these programmes can maintain or even ameliorate the zootechnical performances. The incidence of leg problems can be reduced but sometimes reductions in breast meat yield are found too (Newcombe *et al.*, 1992; Renden *et al.*, 1992; Renden *et al.*, 1993; Classen *et al.*, 1994; Van Harn and Van Middelkoop, 1997). As an alternative for restricting feed intake, sometimes water restriction programmes are used. In the course of this work, however, these subjects will not be further discussed.

5. Compensatory growth

5.1. Description

There is a general consensus that an early growth retardation induces an accelerated growth, known as compensatory growth, which results in final body weights equal or even exceeding that of broilers fed *ad libitum* (Plavnik and Hurwitz, 1985, 1988, 1991). In general, compensatory growth is defined as the abnormally rapid growth relative to age within a breed of an animal after early growth retardation. In Figure 10 a schematic representation of the growth curves of *ad libitum* fed broilers on the one hand and feed restricted broilers on the other hand is given. The two curves show clearly that the early feed restriction induces a significant growth depression, which induces the desired catch–up growth during the last weeks of age. Indeed, sufficient ‘catch-up’ is important to maintain the desired body weights at market age.

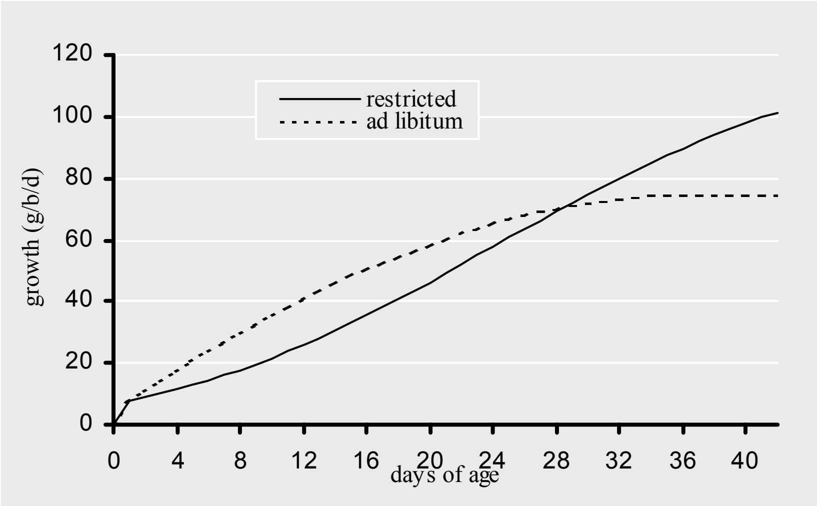

Figure 10 : Comparison of the growth (g/b/d) in function of age between *ad libitum* fed and restricted birds

The mechanisms underlying compensatory growth are not yet fully understood. It is indeed a very complex matter because it involves genetic, physiological, nutritional, metabolic, endocrine and behavioural aspects. However, it is suggested that it is related to a reduced maintenance energy expenditure, increased gut fill and diet digestibility and reduced energy content of the body mass gain (Carstens *et al.*, 1991; Murphy and Loerch, 1994). Indeed, physiological adaptations occur when animals are fed a restricted level of energy intake. Due to reduced physical activity, maintenance requirements are reduced. Moreover, the energy flow is redistributed mainly into activities for maintenance and repair functions while certain energy-wasteful activities, which may not be metabolically essential for growth and maintenance, are reduced. This also results in a reduced basal metabolic rate. Moreover, an increased capacity and slower evacuation of the gastro-intestinal tract (mainly the storage organs) increases the supply of nutrients during the period of feed deprivation.

When refed, the concomitant compensatory growth is characterised by increased intakes and increased efficiency of both energy and protein utilisation due to an accelerated tissue metabolism (Buyse *et al.*, 1996), a reduced maintenance requirement and an activated endocrine status.

Numerous hormones are directly or indirectly involved in the metabolic responses to feed restriction and the subsequent period of refeeding. There are e.g. fast increases in plasma concentrations of insulin (Yambayamba *et al.*, 1996), triiodothyronine (T3) (Nir *et al.*, 1996; Buyse *et al.*, 2000), growth hormone and insulin-like growth factor-I (IGF-I) (Kühn *et al.*, 1996; Buyse *et al.*, 2000) during the refeeding and/or compensatory growth phase. Indeed, these hormones are all known to be regulated by diet and to promote protein accretion and growth rate (Grizard *et al.*, 1999).

5.2. Factors influencing compensatory growth in broiler chickens : literature data

The factors influencing compensatory growth capacity during the period of realimentation include the nature, severity and duration of the undernutrition as well as the age of the start of undernutrition and the degree and pattern of realimentation. Moreover, the time between restriction and slaughter age is obviously determining the ability of the bird to realise compensatory growth. However, also genetic factors such as sex and strain seem to have their impact. All these factors help to explain the rather variable results of feed restriction programmes when considering final body weights. An overview of the main results in literature is given in the Tables 3-7.

5.2.1. Duration and timing of the feed restriction

In general, the longer the period of undernutrition, the more difficult it is for the bird to recover and to compensate for the reduction in weight gain. A feed restriction to 167.4 kJ ME/day starting at the age of 5 days, for 3 or 5 days did not result in any weight gain depression at 54 days of age, whereas a slight depression occurred when this restriction was continued for 7 days (Plavnik and Hurwitz, 1988). These findings are consistent with the results of many other workers (Rosebrough *et al.*, 1986; Plavnik and Hurwitz, 1990; Ballay *et al.*, 1992; Madrigal *et al.*, 1995; Lee and Leeson, 2001).

Even with mild restrictions, prolonged restriction periods induce a significant reduction in final body weights. Mollison *et al.* (1984) restricted feed intake to 90 % of that of the control birds from 7 to 49 days of age, in view of reducing fat deposition and losses due to metabolic losses, and reported a significant lower final body weight (49 d). Even with a restriction of 95 % of the *ad libitum* intake from 5 to 42 days of age, Urdaneta-Rincon and Leeson (2002) found a significant lower final body weight (42 d). Indeed, it seems that restricting feed intake of broilers in the final stages of production allows little or no time to exhibit compensatory growth.

Concerning age at restriction, Plavnik and Hurwitz (1988) reported no difference in overall response when a 7-day restriction period was applied to male broilers at various ages between 3 and 11 days. According to these authors however, for females the restriction should begin before the age of 5 days. Urdaneta-Rincon and Leeson (2002) draw similar conclusions. On the other hand, according to Cristofori *et al.* (1997) no mutual difference in final body weights (42 d of age) were observed when females were fed an amount just satisfying their metabolic

requirements during an early (7 to 21 d) or a late feed restriction (21-35 d). Final weights on both restriction patterns were however significant lower (6 to 8 %) than the control group.
Also the time between the restriction and age of slaughter yield should be taken into account. In the studies of Plavnik and Hurwitz (1985, 1988, 1989) very prolonged trial periods are used (around 56 days of age), which gives the bird the opportunity to recover more easily in comparison with common practice circumstances (42 d or less).

5.2.2. The severity of the feed restriction

During a period of restriction, birds can be fed at, above or below maintenance energy requirements. The more severe the restriction, the greater the initial catch-up growth is, however, the less the ability of the bird to recover completely. Based on the recommendations of Plavnik and Hurwitz (1985), many researchers restricted the birds to a level meeting the maintenance energy requirement. These workers estimated the metabolisable energy requirement for maintenance for male broilers to be 6.3 kJ x $W^{0.67}$ (body weight in grams). However, this maintenance energy must have been overestimated because still a growth of about 2-4 g/day was realised by these birds (Plavnik and Hurwitz, 1985). On the other hand, it is also possible that birds, even being in a negative energy balance, were able to gain weight due to a change in body composition (using fat reserve and deposit more lean tissue) (Yu and Robinson, 1992). Using these restrictions, Plavnik and Hurwitz (1985, 1988, 1989) obtained a sufficient compensatory growth. Lee and Leeson (2001) got even relative heavier birds at 49 days of age when restricting birds to only 3.1 kJ x $W^{0.67}$ for 4 days (imposed at 6 days of age) in a first trial. However, compensation was lacking in the following trials with similar restrictions. Also in own research (Lippens *et al.*, 2002a) Ross male broilers restricted to maintenance energy requirements as short as 4 days starting from day 4 resulted in final body weights (at 42 d of age) being significantly lower (-144 g) in comparison with the *ad libitum* fed group. These findings also confirmed earlier research from Pinchasov and Jensen (1989), Yu *et al.* (1990), Robinson *et al.* (1992) and Palo *et al.* (1995). It is clear that milder restrictions could permit a more realistic recovery (Plavnik and Hurwitz, 1991).
Jones and Farrell (1992a) stated that full body weight recovery might be more consistently realised if the restriction period is subdivided in a number of short restriction periods. By subdividing the period in shorter and less severe periods the bird loses little or no weight, or even gains some weight during the restriction period, which may lead to full recovery more consistently (as a result of improvement in the efficiency of lean tissue deposition and energy

retention) (Jones and Farrell, 1992a). However, varying the period of restriction did not affect growth compensation capacity in the findings of Zubair and Leeson (1994a).
In the Ross Breeders Broiler management manual (1999) an aim for a liveweight reduction of approximately 10-14 % at 14 days of age and 8-12 % at 21 days (as hatched) is proposed when considering a target processing weight between 2 and 2.5 kg.

5.2.3. Nutritional conditions during the period of refeeding

Nutritional conditions during the refeeding period are very important. Still, there is little information available concerning the requirements during this period. Moreover, advises in the consisting literature are sometimes conflicting.
In a study of Plavnik and Hurwitz (1989) the amino acid requirements during the phase of realimentation were re-evaluated. Their calculation models showed a higher requirement for all amino acids during the first two weeks after the restriction. At the end of the trial, however, the response to feed restriction was not significantly modified by dietary protein. On the other hand, these authors could not find any interaction between feed restriction and either energy density or pelleting during the entire period after restriction.
In a more recent study of Jones and Farrell (1992a), the supplementation of the broiler finisher diet with lysine and/or methionine produced non-significant increases in the bodyweight of restricted birds at slaughter although the abdominal fat pad (g/kg bodyweight) was reduced. However, Santoso *et al.* (1995) could not indicate any positive effect of increasing the protein content during 7 days following a skip-a-day programme of 7 days. Acar *et al.* (2001) increased the lysine concentration in the grower and finisher after a feed restriction to 75 % of the metabolisable energy required for normal growth. It was concluded that the increase in lysine did not have any beneficial effect on the final body weight, percentage carcase yield or any of the carcase characteristics with the exception of the *Pectoralis minor* muscle yield, which was increased.
Leeson and Zubair (1997) also concluded that there does not seem to be any advantage to increase the level of protein or lysine during the realimentation of birds previously nutrient-restricted. According to their findings birds are at this time more responsive to energy although this response may be associated with the undesirable trait of increased body fat deposition. However, this study was already terminated at the age of 21 days. As compensatory growth may have been prolonged in the subsequent period, different conclusion might have been found. Indeed, in own research it was found that restricted Ross 308 birds

tended to have a higher final body weight when fed higher protein during realimentation, especially during the last two weeks of age (Lippens and De Groote, 2000). For the Ross 508-line however, only a continued increase in protein after restriction (2-6 weeks of age) induces a non-significantly higher final body weight in comparison with the control.

5.2.4. Genetic factors : sex, strain or line of birds

As males and females differ in growth rate and body fat content (Fisher, 1984; Leenstra, 1986), it has been stated also that male and female broilers react in a different way to feed restrictions. Indeed, according to Plavnik and Hurwitz (1988, 1990, 1991) male broilers have a greater ability to establish compensatory growth in comparison with females. In the research of Deaton (1995), however, both males and females could overcome a weight reduction of 27 and 31 % (feed restriction to 60 % of the *ad libitum*-intake) introduced at 8 to 16 days of age by the age of 48 and 49 days of age, respectively.

Many times, the lack of consistent effects of growth retardation has been attributed to differences in strains of birds used. Cherry *et al.* (1978) stated that faster growing lines exhibited little compensatory growth, while the relatively slower growing ones exhibited considerable ‘catch-up’.

6. Effect of growth retardation and compensatory growth on feed efficiency and body composition

As mentioned above, the effect of growth retardation on the final body weight is rather variable. However, also the effect on feed conversion (FC) is not consistent in literature. With compensatory growth, an improved feed conversion is expected (see above). However, in practice, improvement in feed utilisation is not always found. When considering literature results (Tables 3-7) it can be concluded that often, improved feed conversion is established at the expense of body weight.

As mentioned before, several researchers have determined the proportions of body components of restricted birds at the end of the rearing period (Yu *et al.*, 1990; Fontana *et al.*, 1993; Zubair and Leeson, 1994b), however, there is not much information available about the developmental changes of proportions during the entire growth period. In a study of Govaerts *et al.* (2000), the b-coefficient (cf. 2.2. Allometric growth) of the stomachs was decreased using a quantitative feed restriction (to 80 % of *ad libitum* from day 4 to 11), indicating an

earlier maturation. In a study of Buyse (2001) no effect of the lighting schedule on the b-coefficient of the lungs was found, although an increased proportion was indicated.
Similar results were found for the breast meat, which was confirmed by the findings of Govaerts *et al.* (2000) using feed restrictions. However, the b-coefficient of the thigh using a restriction to 80 % (4-11 d of age) had a tendency to be higher, hence later maturing, in the study of Govaerts *et al.* (2000).
One of the most controversial aspects of growth retardation is the changing effect on abdominal and carcase fat. As fat is a very important aspect of poultry production, a lot of studies examined the effect of feed restriction on fat deposition.
Indeed, next to the fact that high fat depositions are not desirable in terms of energy cost, it also has become a major issue of health concern for the consumer. Excess fat intake has been correlated with obesity, cardiovascular diseases and cancer. Moreover, in recent years, the proportion of broilers used for partitioning and further processing has become more and more important (Figure 11). Also in this context, fat deposition, especially abdominal fat, has become a very important factor as it contributes to higher wastes and costs. On the other hand, a minimum quantity of carcase fat is necessary for an optimal sensory quality because of its positive influence on succulence and taste. The control of lipid accumulation within the cells depends upon the balance between synthesis (lipogenesis) and degradation (lipolysis). In avian species, the liver is the major site for lipogenesis. Besides, the size of the adipose depots depends firstly on the number and secondly on the size of the adipose cells. In most mammals, also human beings, stages in the process of fat depot development are correlated with the age. In the early stage of life, increased cell numbers rather than cell size is the predominant factor. This trend is however reversed in a later stage of life. Moreover, there are two rates of fat deposition, a first, slow one, followed by a faster one (Jones and Farrell, 1992b). According to Cherry *et al.* (1984), this change in rate of deposition may mark the change from the adipocyte hyperplasia (increase in cell number) to the adipocyte hypertrophy (increase in cell size).

Fisher (1984) suggested that feed restriction during the first growth stage of life could reduce hyperplasia, which, at this stage, accounts for most of the growth of the adipose tissue. In this way, total adipose volume at the finishing age could be reduced. Indeed, in the study of Zubair and Leeson (1996b) a significant decrease in fat cell number was found for feed-restricted birds which was still apparent at the age of 42 days. This observation was confirmed by the study of Cartwright *et al.* (1986) but not by Jones and Farrell (1992b) and Zhong *et al.* (1995). According to the results of these latter researchers, the decreased fat content found in the restricted-refed broilers was attributed to a decrease in cell size rather than cell numbers. In a subsequent report from Meluzzi *et al.* (1998), it was concluded that the increase in cell size is significantly suppressed by feed restrictions only if they are applied in the first weeks of life. Late restrictions do not modify the adipocyte size.

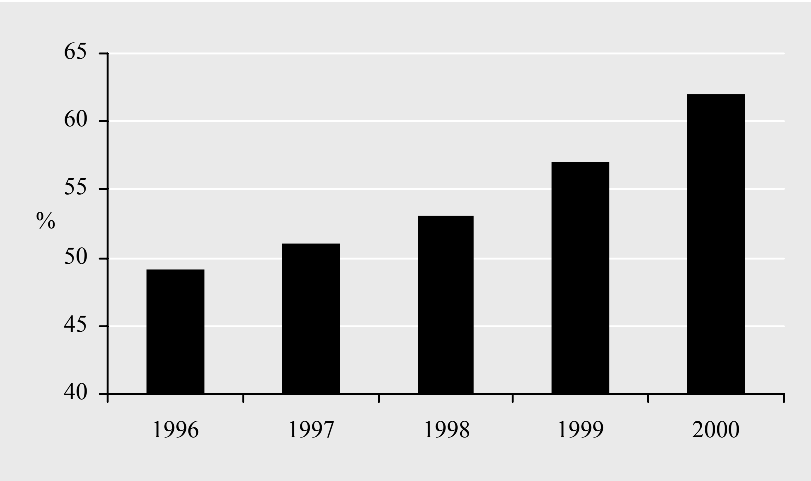

Figure 11 : Evolution of the share of chickens used for partitioning in Belgium (Source : GfK Belgium)

Very interesting results are found by Rosebrough *et al.* (1986) who reported a lowered *in vitro* activity of enzymes involved with hepatic lipogenesis, during the undernutrition, but during refeeding, there was a dramatic increase in such enzyme activities. Total lipogenic capacity increased over 80-fold during the first 2 days following refeeding. After two weeks following the restriction period, the activities declined to levels lower than found with the control birds. This phenomenon can explain the findings of Zubair and Leeson (1996b), in which feed-restricted birds gained about 51 g of fat per bird during the first 5 days of realimentation. For the control groups this was only 26 g during that same period. In this period, a dramatic increase in cell size was established resulting in a similar cell size to their full-fed counterparts. An increase of abdominal fat after refeeding restricted birds, was also found by Cristofori *et al.* (1997) and own research (not published). It is not clear how the short rebound and subsequent decline has to be explained. Cartwright *et al.* (1988) and Cartwright (1991) found a very strong correlation between development of body mass and adipocyte hyperplasia. In other words, as the hyperplasia is delayed by feed restrictions, it is assumed

that birds have the ability to "catch-up" in body fat to a level appropriate for their body mass (Cartwright, 1991; Zubair and Leeson, 1996b).
Interesting are the findings in the study of Buyse (2001) considering the allometric growth of the different fat depots of birds reared in different lighting programs. Using an intermittent lighting schedule, the fat deposition was postponed due to the retardation of the growth. This also contributed to a more efficient feed conversion.
Anyhow, in most cases in literature, a reduction in fat deposition was only established with too severe restrictions resulting in final body weights lower than the control groups (Tables 3-7). Some exceptions are found, in which a reduction in the abdominal/carcase fat content after growth retardation was accompanied with nearly complete recovery of body weight. Still other literature references could not show a clear effect of feed restriction on fat deposition (Tables 3-7).
In addition it seems that fat deposition rate in between birds of a same treatment also, knows an extremely high variability (own research, not published). This also may be one of the reasons for the sometimes conflicting results found in literature.
Next to fat content, but far less studied in literature, also slaughter yield and the percentage of the different cut-up parts are of major importance. Especially the percentage breast meat, because it is economically the most valuable part of the carcase, needs much attention in the study of feed restriction programmes.
Dutch researchers warned for losses in slaughter yield and/or breast meat percentage when using restriction programmes (Van Harn and Fabri, 1995; Van Middelkoop, 1997) although zootechnical performances were not always impaired significantly (Van Harn and Van Middelkoop, 1998). On the other hand, many researchers could not indicate any negative effect on dressing percentage or breast meat yield (Pinchasov and Jensen, 1989; Plavnik and Hurwitz, 1990; Yu *et al.*, 1990; Leeson *et al.*, 1991; Carter *et al.*, 1994; Classen *et al.*, 1994; Zubair and Leeson, 1994a; Palo *et al.*, 1995; Acar *et al.*, 2001). In a very recent study of Lee and Leeson (2001), some changing results are found. When male birds are restricted to 6.28 kJ ME x $BW^{0.67}$ (body weight in gram) from day 7 until day 11, a significant increase in breast meat % was found (20.9 and 21.7 %, for the controls and the restricted birds respectively). Final body weights of these restricted birds too, were significant heavier in comparison with the *ad libitum* group. However, when the diet was diluted with 50 % oat hulls from 7 to 14 days, both final body weights and breast meat percentages were lowered. This observation is also confirmed by the findings of Newcombe *et al.* (1992); Leeson *et al.* (1999); McGovern *et al.* (1999) and Urdaneta-Rincon and Leeson (2002). In a study of Govaerts *et al.* (2000), the

proportional breast weight of restricted birds was lower short after the restriction, but these effects disappeared at slaughter age. In a study of Buyse (2001) however, an intermittent lighting schedule (1L:3D) had no significant effect on the allometric coefficient but, during the entire growth trajectory, the proportion of breast meat tended to be higher for the control birds (23L/1D). Generally spoken, it seems that when the catch-up in final body weight is complete, no significant losses in breast meat should be expected. Only when significant higher final body weights are realised, increases in breast meat percentages can be found. The influence of feed restriction on carcase yield and breast meat percentage is further discussed in this thesis.

7. Effect of growth retardation and compensatory growth on meat quality

Only little literature information is available with regard to the effect of feed restriction and compensatory growth on meat quality. Meat quality can be described by colour, pH, water holding capacity/cooking losses, tenderness and taste.

As in adipocyte tissue, also in muscle growth, hyperplasia and hypertrophy are the determinants of muscle mass. The number of fibres is, in the majority of species, fixed at birth (Grizard *et al.*, 1999). The balance between the amount of muscle protein synthesised and the amount of muscle protein degraded determines muscle size. However, according to Koohmaraie *et al.* (2002), changes in protein synthesis does not affect meat tenderness. They concluded that, of all the possible mechanisms of increasing muscle protein accretion, only the mechanism that involves suppression of protein degradation results in decreased meat tenderness. Based on research of the proteolytic capacity of different broiler strains with different growth rates, Dransfield and Sosnicki (1999) indeed suggested, that the increased growth and muscle mass in modern lines could be largely governed by reduced protein catabolism.

In general, according to these authors, faster growth may be correlated with morphological abnormalities, larger fibre diameters, a higher proportion of glycolytic fibres and a lower proteolytic potential in the muscles. If these considerations were correct, a faster development of the post-mortem rigor mortis would occur. This process might increase the incidence of paler and tougher meat, with a reduced water holding capacity.

Indeed, it has been known that muscle fibres can be physiologically differentiated for fast and slow contraction and for aerobic and anaerobic metabolism. There are fibres which are specialised for aerobic metabolism, having many mitochondria (type I) and fibres which are specialised for anaerobic metabolism, having few mitochondria and high glycogen activity (type IIb). In addition, there are some fibres that exhibit both conditions so that they are able to obtain energy for muscle contraction both aerobically and anaerobically (type IIa). The breast muscle of Galliformes is entirely composed of the type II fibres, whereas the leg muscles exist of a mixture of all three types (Rémignon *et al.*, 1996). Indeed, research shows that, due to selection for growth rate and breast meat yield, a shift from type I towards more type IIb muscle fibres has occured, which has a major impact on post mortem energy metabolism and thus, on meat quality (Decuypere *et al.*, 2000).

Le Bihan-Duval and Berri (1999) could confirm this statement of Dransfield and Sosnicki (1999) by comparing selected lines (increased body weight and breast meat yield) with its control line. Also Mitchell (1997) and Velleman *et al.* (2003), reported a link between growth rate and an increased susceptibility to muscle damage. According to this author, the incidence of muscle disorders in poultry, including meat toughness and pale, soft, exudative meat (PSE) especially in turkeys, increases. According to studies of Barbut (1997) and Sams *et al.* (1999), the occurrence of PSE in broiler chickens ranges from 0 to 28 %, depending on the flock. In a study of Berri *et al.* (2001), however, there was no support for the idea that selection had a negative effect on meat quality (colour, drip loss), despite the evidence of modified breast metabolism (pH, metabolic enzyme activities).

It is, however, not clear from literature if the modification of the growth curve by nutritional manipulations as mentioned here, has significant effects on the meat quality. In a study of Close (1997) it was concluded that *ad libitum* fed pigs produced more tender meat than pigs fed to only 80 % of *ad libitum* intake. Ballard *et al.* (1988) could not indicate statistically significant differences in muscle calcium-activated neutral proteinase (calpain) and its specific inhibitor (a system thought to be implicated in myofibrillar catabolism) in chickens grown at different rates in response to graded levels of dietary protein. Only a trend of increased activity was found with increased growth rate and muscle weight.

A study of Fujimura *et al.* (1997) mentioned that, in the meat extract of restricted chicks, some taste-active components (free glutamine and some amino acids) tended to decrease in comparison with *ad libitum* fed birds.

The influence of quantitative and qualitative feed restrictions on subsequent meat quality (pH_{24}, colour parameters, moisture, drip, cooking losses, shear force) is examined in this thesis.

8. Feed restriction and animal welfare

Several parameters can be used as a measurement of animal welfare. For the treated subject – feed restriction- results are equivocal. As mentioned before, certain feeding programmes can reduce the incidence of leg disorders and metabolic diseases and thus the associated pain and suffering. In this way, the welfare of feed restricted broilers is enhanced. On the other hand, there are some reports mentioning a lowered immunity when birds are restricted (Kondo *et al.*, 1988; Payne *et al.*, 1990; Afzal *et al.*, 1999). This conclusion was based on the findings that after restrictions lowered concentrations of among others, lymphocytes and macrophages are measured. As a result, these birds may become more susceptible to various pathogens. These findings, however, are contrary to the results of Ballay *et al.* (1992), who found a less severe response to an *E. coli* inoculation when birds were feed restricted.

According to Hocking *et al.* (1993), feed-restricted broiler breeders spent a large proportion of their time scratching and pecking on the litter compared with their *ad libitum* fed counter parts. An increased plasma concentration of corticosterone, as found by Hocking *et al.* (1993), could indicate a higher level of susceptibility to stress. Also an increase in the heterophyl:lymphocyte ratio, a measure for chronic stress, was found by these authors. However, in the research of Maxwell *et al.* (1990), this latter ratio was not significantly altered by feed restriction.

Hocking *et al.* (1996) concluded, however, that the limit of an acceptable stress is only reached at a 75 % reduction in body weight. This conclusion is based on the considerations that birds showed a normal physiological response to this kind of feed restriction without any apparent changes in pathology and immunological function. However, using a more severe standard of acceptability, e.g. the levels of basophils and plasma corticosterone, they concluded that this level of restriction might indeed be considered to be unacceptable.

In general, based on response parameters such as the higher incidence of muscle tissue-damage and increased fearfulness, the higher mortality and incidence of leg disorders, Hocking *et al.* (1996) could conclude that moderate restriction programmes, especially in broilers, might be beneficial thereby not inducing poor welfare.

Table 3 : Overview of the main results of trials on feed restriction in literature (I) [1]

authors	year	restriction	end of trial	final body weight	FC	Carcase/abd. Fat	breast meat	skeletal/ metabolic diseases
Acar *et al.*	1995	qn 75 % 4-11d or 7-14d	49 d	-	0	-	-	+
Acar *et al.*	2001	qn 75 % 4-11d	56 d	-	0	-	0	+
Albers *et al.*	1990	qn 73 % 7-21d; ql low energy/low fat	57 (60) d	0	+	X	x	+
Arce *et al.*	1992	qn 90 % cont. skip a day starter	53 d 51/56 d	- 0	0 0	X X	x x	+ +
Attia *et al.*	1993	qn 50 % 7-21d	49 d	x	x	0	x	x
Ballay *et al.*	1992	alternate-day (≠ periods)	39 d	-/0	0	0/+	-/0	x
Beane *et al.*	1979	qn 85 % 15-42d	56 d	-	+	-	x	x
Bowes *et al.*	1988	qn 75 % 5-39d	39 d	-	0	X	x	+
Bruno *et al.*	2000	qn 40 % 7-14d	42 d	-	x	X	x	x
Cabel & Waldroup	1990	Qn fasting, maintenance level 5-11 or 17d dilution (50 % sand) 5-11 or 17d	49 d	-/0	0/+	0/+	x	x
Calvert *et al.*	1987	qn 167 kJ/d 6-12d	56 d	0	+	X	x	x
Carter *et al.*	1994	ql low prot. 7-14d	49 d	0	0	X	0	+
Cartwright *et al.*	1986	qn maintenance 6-12/18d	49 d	-/0	x	+	x	x
Cherry *et al.*	1978	ql low dens. 1-28d	56 d	+/-	-	+/-	x	x
Classen *et al.*	1994	ql low density diets (≠ periods)	35 d	-/0	-/0	X	0	0/+
Cristofori *et al.*	1997	qn 6.3 x $BW^{0.75}$kJ/d[2] 7-21 and 21-35d skip a day	49 d	-	+	0	x	x

[1] abbr.: qn = quantitative restriction; ql = qualitative restriction; x = not determined; 0 = no effect, + = positive effect, - = negative effect
[2] BW in gram

Table 4 : Overview of the main results of trials on feed restriction in literature (II)[3]

authors	year	restriction	end of trial	final body weight	FC	carcase/abd. fat	breast meat %	skeletal/ metabolic diseases
Deaton *et al.*	1973	ql low energy 0-28d	56 d	0	0	-/0	x	x
Deaton	1995	qn 60 to 90 % 7-14 and 8-16d	41/48/49 d	-/0	0/+	0	x	0
Dozier *et al.*	2002	skip a day : 2, 4 or 6d from d 8	54 d	0	0	0	x	0
Fancher and Jensen	1988	ql chem. 0-14; 0-21; 21-42d	42 d	-/0	-/0	0/+	x	x
Fontana *et al.*	1992	qn 167 kJ/d d4-9;4-10 or 4-11d	49 d	-	+	x	x	+
Fontana *et al.*	1993	qn 167 kJ/d 4-10 or 4-11d	49 d	x	x	0	x	x
Gonzales *et al.*	1998	qn 80 % 8-21d	42 d	-	+	x	x	+
Griffiths *et al.*	1977	ql low energy 0-7;0-14; 0-21 or 0-28d	56 d	0	0	0	x	x
Huyghebaert *et al.*	1991	qn 7.5 x $BW^{0.67}$ kJ/d[4] 6-11 d ql low energy - low energy/protein (-4.8 %/ –21%)	45/52 d	- -	+ 0	0/+ 0	0 0	x
Jones and Farrell	1992a	qn 20 % (≠ periods); 3.1x $BW^{0.67}$ kJ /d[4] 7-10d dilution (65 or 60 % rice hulls) 2x2d from d7	49 d	0	+/-	0/+	x	x
Jones and Farrell	1992b	qn 3.1 x $BW^{0.67}$ kJ/d[4] 7-10d or 7-12d	70 d	0/-	0	0/+	x	x
Katanbaf *et al.*	1988	skip-a-day 6-28d	42 d	-	-	0	-	x
Lee and Leeson	2001	qn (≠ periods, ≠ levels) dilution (50 % oat hulls) (≠ periods, ≠ levels)	49 d	-/+ -/0	0/+ 0	0 0	0/+ -/0	x
Leeson *et al.*	1991	dilution (25, 40, 55% rice hulls) 4-11d	42/56 d	0	0	0/+	0	x
Leeson *et al.*	1999	ql low protein/energy 21-49d	70 d	-/0	-/0	0/+	-/0	0/+

[3] abbr.: qn = quantitative restriction; ql = qualitative restriction; x = not determined; 0 = no effect, + = positive effect, - = negative effect
[4] BW in gram

Table 5 : Overview of the main results of trials on feed restriction in literature (III)[5]

authors	year	restriction	end of trial	final body weight	FC	Carcase/abd. Fat	breast meat %	skeletal/ metabolic diseases
Leeson and Zubair	1997	qn 50 % 6-12d dilution (50 % oat hulls) 6-12d	21 d	-	+	0	x	x
Leterrier and Constatin	1996	dilution (49 % wheat bran): 1-21 or 1-42 d	42 d	-/0	-/0	x	x	+
Leterrier *et al.*	1998	ql low energy continued	42/46 d	-	-	x	x	+
Madrigal *et al.*	1995	dilution (up to 60 % rice bran) 3-10; 7-14d	49/56 d	0	0	x	x	-/+
Marks	1979	qn low protein 0-14d; 0-56d	56 d	-/0	0	x	x	x
McGovern *et al.*	1999	qn (18 g of feed/d) 7-16d	40 d	-	+	+	-	+
Meluzzi *et al.*	1995	ql (Na-deficiency) 7-14d ql low density 1-14d	49 d	0 -	+ +	0/+ 0	x	x
Meluzzi *et al.*	1998	qn 6.3 x $BW^{0.75}$kJ/d[6] 7-21d or 21-35d skip a day 7-28d	49 d	0 -	x	0	x	x
Mollison *et al.*	1984	qn 90 % 7-49d ql fat restr. 0-7d	49 d	- 0	+ 0	+ -	x	0 0
Moran	1979	ql protein restr. 14-35d	49/56 d	0	0	-	0	x
Mudrić *et al.*	1994	qn 6.3 x $BW^{0.67}$ kJ/d[6] 6d (cont. /discon.) from d7	42 d	-	0	0/+	x	x
Newcombe *et al.*	1992	qn 9.414 or 6.276 x $BW^{0.67}$ kJ/d[6] 5-11d	49 d	-	0	+/0	-	x
Palo *et al.*	1995	qn 3.10 (6.28) x $BW^{0.67}$kJ/d[6] 11-14d (7-14d)	48 d	-	+	0	0	x
Pinchasov and Jensen	1989	qn 146 (151) or 251 (272) kJ/d 7-14d ql chem. 7-14d	49 d	-/0	+	0	0	x

[5] abbr.: qn = quantitative restriction; ql = qualitative restriction; x = not determined; 0 = no effect, + = positive effect, - = negative effect
[6] BW in gram

Table 6 : Overview of the main results of trials on feed restriction in literature (IV)[7]

authors	year	restriction	end of trial	final body weight	FC	carcase/abd. fat	breast meat %	skeletal/ metabolic diseases
Pinchasov *et al.*	1985	alternate days 14-56 or 14-83d	83 d	-	+/0	+	x	x
Plavnik and Hurwitz	1985	qn 125.5 up to 188 kJ/d (≠ periods)	56/63 d	-/0	0/+	+	x	x
Plavnik and Hurwitz	1988	qn 6.3x$BW^{0.67}$ kJ/d[8] (≠ periods)	54/56/59 d	-/0	+	+	x	x
Plavnik and Hurwitz	1989	qn 6.3x$BW^{0.67}$ kJ/d[8] 6-12 or 6-13d	51/55/57 d	0	+	+	x	x
Plavnik and Hurwitz	1990	ql low prot., low Na 6-12 or 8-14d	56 d	-/0	0/+	+	0	x
Plavnik and Hurwitz	1991	qn 0 to 75 % of growth, 7-14 or 6-10d	50/56 d	-/+	0/+	+	-/0	x
Plavnik and Yahav	1998	qn 60 % of normal growth rate 6-12d	56 d	0	0	+	+	x
Plavnik *et al.*	1986	qn 6.3x$BW^{0.67}$ kJ/d[8] from d5/6 for 6 or 12 d	56 d	-/+	+	+	x	x
Proudfoot *et al.*	1983	feed denial 8 or 12h/d from 8-21d or 15-28d	49 d	-/0	0	x	x	0
Robinson *et al.*	1992	qn 6.3x$BW^{0.67}$kJ/d[8] (≠ periods); skip a day dilution (50 % oat hulls) 7-14d	63 d	-/0	x	0/+	x	0/+
Rosebrough *et al.*	1986	qn 6.3x$BW^{0.67}$ kJ/d[8] 6-12; 5-11 or 6-18d	27/54 d	-/0	x	0/+	x	x
Roth *et al.*	1993	qn 50, 60, 70 % : 5-19d/26d/33d; 65 % 5-18d or 12-25d	40 (42) d	-/0	0/+	x	x	x
Saleh *et al.*	1996	qn 20, 30, 40 % : d8,9,12 and 13	49 d	-/0	0	0/+	+	0/+
Santoso *et al.*	1995	skip-a-day 7-14d	56 d	0	0	+	x	x
Scheideler and Baughman	1993	qn 50 % 6-14d or 65 % 8-14d	42/63 d	-/0	0/+	0	0/+	0/+
Summers *et al.*	1990	qn 50 % (70 %) or +15% alpha floc 7-14d	41 d	-/0	-/0	0	x	x

[7] abbr.: qn = quantitative restriction; ql = qualitative restriction; x = not determined; 0 = no effect, + = positive effect, - = negative effect
[8] BW in gram

Table 7 : Overview of the main results of trials on feed restriction in literature (V)[9]

authors	year	restriction	end of trial	final body weight	FC	carcase/abd. fat	breast meat %	skeletal/ metabolic diseases
Susbilla *et al.*	1994	50 % or 75 % 5-11 d	39 d	0	0/+	0	0	x
Urdaneta-Rincon and Leeson	2002	qn 85 to 95 % d 5 or d 14; ≠ durations	42 d	-/0	+	0	-	0/+
Van Harn and Fabri	1995	qn (in function of bodyweight)15-42d	42 d	-	0	+	-	+
Van Harn and Van Middelkoop	1998	qn low protein 8-14d or 15-21d ql 86 % 8-14d	41 d	0	0	0	-/0	0
Van Middelkoop	1997	qn (in function of bodyweight)14-42d	42d	-	0	x	-	+
Washburn	1990	qn 3-28d; 3-49d	49 d	-	+	+	x	x
Yu *et al.*	1990	qn 4.184 $BW^{0.67}$kJ/d[10] 8-14 d	56 d	-	0	0	0	x
Zhong *et al.*	1995	qn 6.2x$BW^{0.67}$ kJ/d[10] 7-12d	49/56 d	-/0	+	0/+	x	x
Zubair and Leeson	1993	qn 50 % 6-12d	42d	-	+	0	x	x
Zubair and Leeson	1994a	dilution (50 % oat hulls) 6-12d cont./discont.	49 d	0/+	0/+	-/0	0	x
Zubair and Leeson	1994b	qn 50 % 6-12d	21 d	-	+	x	x	x
Zubair and Leeson	1996b	qn 50 % 6-12d	42 d	-	+	0	x	x

[9] abbr.: qn = quantitative restriction; ql = qualitative restriction; x = not determined; 0 = no effect, + = positive effect, - = negative effect
[10] BW in gram

Chapter 3

GENERAL MATERIALS AND METHODS

1. Introduction

Materials and methods are kept as constant as possible throughout the different trials. The following materials and methods apply for all trials unless otherwise stated in the 'materials and methods'-section of the respective trials.

2. Diets

The diets used in each trial are given in the respective chapters. Nutrient requirements are based on NRC- (NRC 1994) and CVB- (CVB 1997) recommendations. The ideal amino acid profile (Lippens et al, 1997; Mack *et al.*, 1999) was established although shifts are generally seen possible due to least cost formulation. In all diets a vitamin/mineral premix was used which provided the following quantities (mg/kg of diet): retinol, 4.05; cholecalciferol, 0.05; tocopherol, 13.5; menadione, 2.25; thiamin, 1; choline, 375; riboflavin, 5.4; panthothenic acid, 13.5; pyridoxine, 1.1, cyanocobalamin, 0.01; nicotonic acid, 40; biotin, 0.15; I, 2.1; Co, 1.4; Se, 0.43; Cu, 7.2; Mn, 86; Zn, 57; Fe, 65; Mg, 110.

Pellet quality (Chapter 9) was measured sieving a sample of the respective diets. The procedure consisted of intermittent (5 sec on – 0.5 sec off) sieving samples of 500 g for 10 min through a set of 6 steel sieves (Fritsch-analysette). The respective sieve openings were 4 mm, 2 mm, 1 mm, 0.5 mm, 0.25 mm and 0.125 mm.

3. Birds and housing

Two different strains of commercial broiler lines are used in the different trials of this work. It concerns Hybro G (Chapters 4 and 5) and two lines of Ross-broilers. The Ross 208 or 308 (replacing the 208-line completely since September 2000) is the commonly used commercial line of Ross. The Ross 508-line used in most of the following trials, however, is already genetically selected for a lower initial growth in view of reducing metabolic diseases. For this line the highest growth increase takes place around day 26, while for the standard line Ross 308 a maximum is found around 21 days of age (Ross Breeders brochure, 2000). Ross 508 broilers also are characterised by a significantly higher breast meat percentage. As it is interesting to know to which extent the modified line can still have an advantage of additional 'feed-regulated' growth retardation, intensive research on this line is represented in this work.

Birds were kept in floor pens on chopped straw with an available surface of 6.3 (large pen) or 2.1 m^2 (small pen) (Figure 12). Stocking density was 16 birds per m^2. A conventional lighting schedule of 23 h of light and 1 h of darkness was used (except for the trial in Chapter 7). In most trials a standard temperature schedule was used. Mean environmental temperature was 30 °C during the first 3 d. From d 3 onwards ambient temperature was set at 28 °C to decrease with 1 degree every 3 d until the temperature of 21 °C was reached at d 21. The 21 °C was maintained until the last day of the trial. Water was freely available to all birds. Each growth trial was carried out during 42 d.

4. Response parameters

4.1. Zootechnical parameters

In the Chapters 4 and 5, male and female birds were reared separately. From Chapter 6 on, it was decided to use a mixed sex flock in accordance with practice. One day-old broilers were placed unsexed to avoid any disturbance of the chickens at that young age. It is known that cloaca sexing can be a major factor of stress. If growth is impaired at this stage of life, no good conclusions can be drawn for the subsequent growth. Moreover, there were some indications in other trials that both lines have different early stress susceptibility (unpublished data). However, sex ratios were determined at the end of the trials, thereby taking into account the sex of dead and removed birds. Body weights were recalculated to a 50/50-ratio (BW_{corr}= (BW_{males}+$BW_{females}$)/2). The correction factor applied to recalculate for differences in feed intake between males and females (FI_{males}=1.144 $FI_{females}$) was based on a theoretical value as mentioned in the manual guide of Ross Breeders. As this correction factor was confirmed by research results (Lippens *et al.*, 2000), it seemed a suitable approach to further eliminate any influence of differences in sex ratio.

All chickens were weighed weekly (except for the trial in Chapter 4) to determine body weight, weight gain per bird and uniformity. Uniformity was calculated as the percentage of birds between ± 20 (10 %) of the mean body weight of the flock. Twenty percent was used in the trials of Chapter 4 and 5. The 10 % was used in the subsequent trials. Results were corrected for the differences in number of males and females per pen, when using mixed sexes, according to the formula U_{corr}= (U_{males}+$U_{females}$)/2.

Feed consumed was recorded daily for all pens. Feed conversion was defined as the ratio between feed intake and weight gain. Dead chickens were removed daily and an autopsy was done to check for indications of SDS or ascites. Birds with visible leg problems were also removed and recorded daily. The sum of death and removed birds is indicated as 'total mortality' in the different trials.

4.2. Preparation for carcase and meat quality

At the end of the experiment (d 42), 12 birds from the large pens and 4 birds from the small pens (12 % of the flocks) with body weights close to the pen average were selected (except for the trial in Chapter 6). When mixed sexes were used in the trial, half of the selected birds were males, the other half females. They were weighed individually after 16 h of fasting. At 43 d the chickens were slaughtered in a local slaughterhouse. The eviscerated carcases (liver, lungs, heart, digestive tract, neck and surrounding skin removed) were chilled in cold air of 2 °C for 24 h. The carcase yield was determined as the weight of the eviscerated carcass relative to the empty live body weight. The abdominal fat content can be defined as the fat surrounding the gizzard and extending within the ischium and surrounding the bursa of Fabricius, cloaca and adjacent abdominal muscles relative to the empty live body weight. After cooling, the carcases were cut into breast, thighs, drumsticks, wings and bones + skin according to a standardised procedure (Uijttenboogaart and Gerrits, 1982) (Figure 13). The weights of these parts relative to the eviscerated carcase weight were determined.

4.3. Meat quality parameters

To examine the meat quality (trials Chapter 4 and 5), 3 breasts (*pectoralis* muscle) per treatment were used. The pH_{24} was determined 24 h after slaughter.
Meat colour was measured on the outer surface of the *pectoralis* with a spectrocolorimeter (LABSCAN II with illuminant D65 (daylight) and 10 Degree Observer) (Figure 14). Three colour parameters were generated : L (lightness), a (redness) and b (yellowness).
One half of each breast was ground with a Ultra-Turrax homogeniser for 2 s. A sample of 300 mg was placed on a filter paper between 2 glass plates. After using a force of 1 kg to press the sample for 5 min, the filter paper (+ moisture) was weighed. The loss of moisture under pressure, as a measure for water holding capacity (WHC), was defined as the ratio between weight of the moisture and the weight of the sample x 100.
The other half of each breast was frozen after vacuum sealing in impermeable bags 24 h after slaughter. The samples were stored at < -20 °C for 2.5 weeks. While thawing, the samples

were kept at 2 °C for 19 h and the drip loss was measured. Thereafter, they were cooked in a water-bath at 75 °C for 50 min (40 min) and cooled under running water for another 50 min (40 min). The following parameters were measured:

$$\text{drip} = \frac{\text{fresh weight - weight after thawing}}{\text{fresh weight}} \times 100$$

$$\text{cooking losses} = \frac{\text{weight before cooking - weight after cooking}}{\text{weight before cooking}} \times 100$$

Shear force was determined with a Warner-Blatzer shear with a down speed of 20 cm/min (Figure 15). The ‘peak’-force is expressed in N and used as a measure for tenderness of the chicken meat.

4.4. Carcase composition

At 43 d also, a representative number of chickens (see each trial) with body weights close to the pen average were killed by cervical dislocation after electrical stunning. The chickens were frozen, minced and mixed, freeze-dried and homogenised. Lipid and protein content of the whole bird (including feathers) were determined on pooled samples (per pen). These contents were determined respectively by Soxhlet-extraction with petroleum-ether (Publication European Communities n° L257/15) and the Kjeldahl procedure (Publication European Communities n° L179/9).

5. Statistical analysis

The data were analysed using a linear model analysis of variance (ANOVA) (Statistica 5.0, 1995). LSD multiple range tests identified separated means at the level of 5 % probability. Non-significant interactions are not presented in the tables of results. All percentage data were converted to arcsines prior to analysis.

Figure 12 : The 'floor-pen' housing of the birds

Figure 13 : Cut-up parts of the chicken carcase according to the standardised procedure of Uijttenboogaert and Gerrits (1982)

Figure 14 : Labscan II

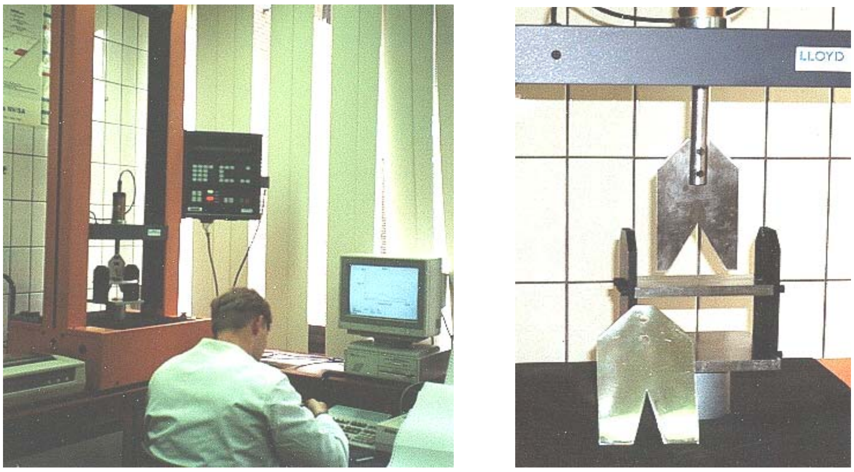

Figure 15 : Warner-Blatzer shear force measurement

The weekly-recorded body weights were fitted to a Gompertz equation. This made it possible to estimate daily body weights and daily growth parameters. The following equation was used:

$$W_t = W_0 \, e^{(L/K)(1-e^{-Kt})}$$

with: W_t : body weight at time t; W_0 : initial body weight (at t = 0); L : slope of the growth curve at time t = 0 or the initial specific growth rate; K : rate of exponential decay of the initial specific growth rate L, which measures the rate of decline in the growth rate.

In the following chapters, growth curves are only visualised for the last weeks of the trial. This makes it easier to have a clear view on the time and magnitude of the compensatory growth for the different treatments. However, in all cases, equations mentioned consider the entire growth period (1-42 days of age).

Chapter 4

QUANTITATIVE FEED RESTRICTION OF BROILER CHICKENS

Adapted from :

Lippens, M., Room, G., De Groote, G. & Decuypere, E. (2000). Early and temporary quantitative food restriction of broiler chickens. 1. Effects on performance characteristics, mortality and meat quality. British Poultry Science 41 : 343-354.

ABSTRACT

1. An experiment was conducted with broiler chickens to determine the effects of different early feed restrictions, strain (Ross 508 and Hybro G) and sex on performance, mortality, carcase composition and meat characteristics.
2. Birds were restricted to 80 % or 90 % of *ad libitum* intake for 4 d (80 % - 4 d-group and 90 % - 4 d-group, respectively) or 80 % for 8 d (80 % - 8 d-group). All restrictions started on d 4. After the periods of restriction, all birds were fed *ad libitum*.
3. Only the 90 % - 4 d-group reached a final body weight not significantly different from, but lower than, the *ad libitum* group. The other restrictions were too severe to allow a sufficient 'catch-up'.
4. No significant differences in feed conversion and total carcase lipid content between groups were observed. Abdominal fat showed a tendency to increase due to the restrictions induced.
5. There was a slight trend towards a reduced mortality and of 'sudden death syndrome' but no clear effect of feed restriction on number of chickens removed with leg problems. There was no significant decrease in uniformity of the flocks due to restriction.
6. The group 80 % - 8 d had a significant lower yield percentage. Cut-up parts and meat quality were not changed by restriction. Ross birds had a significantly higher proportion of breast meat than Hybro chickens. Meat of female chickens seemed to be paler than of males, possibly because of the higher proportion of carcase lipid.
7. Feed restriction did not always give good results. However, a mild restriction (90 % for 4 d) may offer some economic advantages over an *ad libitum* feeding regimen, mainly by reducing mortality.

1. Introduction

Genetic selection in meat-type chickens has provided the industry with flocks which reach the target slaughter weight in a shorter period of time. Research shows that the improved growth rate results from a large increase in early postnatal growth rate (Ricklefs, 1985). However, some unfavourable selection responses have also occurred. These modern meat-type broilers show an increased fat deposition, a higher incidence of leg problems and a greater susceptibility to metabolic diseases such as 'sudden death syndrome' (SDS) and ascites.

One approach in controlling these negative selection responses is to restrict growth in the early stage of life. Studies of early feed restriction resulted in better feed utilisation and reduced carcase fat content without a reduction in final body weight (Plavnik and Hurwitz, 1985, 1988, 1991). Success in reducing body fat deposition however was not achieved by a number of other workers who also used early feed restriction strategies (Scheideler and Baughman, 1993; Deaton, 1995; Zubair and Leeson, 1996b; Cristofori *et al.*, 1997) .

A slower growth rate in the early stage of life can reduce leg problems and total mortality (Robinson *et al.*, 1992, Saleh *et al.*, 1996). Indeed, according to Robinson *et al.* (1992) and Carter *et al.* (1994) temporary feed restriction reduced the incidence of skeletal disorders. Also, metabolic diseases can be reduced (Bowes *et al.*, 1988; Albers *et al.*, 1990; Arce *et al.*, 1992; Fontana *et al.*, 1992; Classen, 1994; Van Harn and Fabri, 1995; Van Middelkoop, 1997; Gonzales *et al.*, 1998; McGovern *et al.*, 1999; Acar *et al.*, 1995, 2001; Urdaneta-Rincon and Leeson, 2002). Other studies (Scheideler and Baughman, 1993; Deaton, 1995; Madrigal *et al.*, 1995) could not confirm these results.

The objective of this work was to study the response of two different strains of broilers to different degrees of feed restrictions at an early age. The effects on performance, compensatory growth, mortality, carcase composition and meat quality were investigated.

2. Materials and methods

2.1. Experimental design

A 3-factorial experiment (4 x 2 x 2) was set up to investigate the effect of different quantitative feed restrictions on two strains of meat chickens. Both sexes were used in the trial. The experiment was conducted with 1596 Ross 508 and 1596 Hybro G day-old chicks obtained from a local hatchery.

Each dietary treatment, per strain and per sex, had three replicates consisting of two large pens each containing 100 birds and of one small pen containing 33 birds, except for treatment 90 % - 4 d, which had only three replicates of 33 birds. Mean daily feed consumption per bird for each strain and each sex was determined from the 4 x 3 pens not restricted (*ad libitum* groups) and was assumed to be representative of the *ad libitum* intake. The birds of the 80 % - 4 d-group received, from d 4 until d 7, 80 % of the determined *ad libitum* intake of the previous 24 h. During the same period, chickens of the 90 % - 4 d were restricted by 10 %. A third treatment (80 % - 8 d) consisted of a restriction to 80 % of *ad libitum* from d 4 until d 11.

2.2. Diets

Except for the duration of the feed restrictions, the birds were fed *ad libitum*. A starter diet with 211 g/kg CP and 12.42 MJ apparent metabolisable energy (broilers; CVB 1997)/kg (AME_n) was given until 14 d of age. From d 15 until d 42 a grower diet with 209 g/kg CP and 12.85 MJ AME_n/kg was offered. For the ingredient composition and the calculated chemical analysis see Table 8.

2.3. Response parameters

General response parameters are described in Chapter 3. All chickens were weighed individually at d 3, 14, 28 and 42 and per pen at d 8, 21 and 35 to determine body weight, weight gain per bird and uniformity. At 43 d, 9 chickens from the large pens and 3 from the small pens were used to determine lipid and protein contents.

3. Results

3.1. Performance

Results of feed intake, body weight, gain and FC at 8 d of age are shown in Table 9. Feed restriction to 80 % or 90 % of *ad libitum* for 4 d significantly decreased body weight at 8 d. Body weight at the end of the restriction had decreased by 9 % and 4 % respectively. At this age the treatment 80 % - 8 d was still proceeding and can not be considered.

The daily body weight gain of birds submitted to the feed restriction was significantly lower when compared to the birds fed *ad libitum*. The retardation of growth was more pronounced for the 80 %-restricted birds. Feed conversion was significantly improved by restriction. The more restricted the more efficiently the feed was converted during this early stage of life.

At the age of 8 d the body weight of Ross chicks was significantly lower than that of the Hybro chicks. Also, the initial body weight at d 0 differed significantly (38.1 g and 43.5 g respectively). These low body weights for the Ross chicks were accompanied with a lower daily weight gain during the first 8 d and a significantly lower feed intake. Indeed, low juvenile growth is typical for the Ross 508 line. FC was not affected by strain.

At this stage no influences of sex on performance were observed. However, a significant interaction between strain and sex at 8 d of age was noted for body weight and body weight gain. This interaction was due to the fact that Ross females were heavier than Ross males whereas the opposite was true for the Hybro chicks. The interactions found for the FC mainly concerned the 80 % - 8 d-treatment, which was still going on at this stage, so they are not considered here.

At 21 d, birds of the 90 % - 4 d-treatment had a body weight similar to the control birds (Table 10).

Table 8 : Ingredient composition and calculated nutrient composition of the diets (g/kg, unless otherwise stated)

Ingredients	**starter (0-14 d)[1]**	**grower (15-42 d)[2]**
Wheat	400.0	500.0
Soybeans (full fat)		153.9
Soybean meal (44 % CP)	253.8	
Soybean meal (48 % CP)		98.6
Yellow corn	190.4	100.0
Animal fat	70.0	50.0
Meat meal (58 % CP, 14 % CF)	50.0	60.0
Tapioca		5.8
Dicalc. phosph. .2H_2O	13.4	10.7
Limestone	3.54	3.97
Sodium chloride	2.28	2.89
Sodium bicarbonate	1.43	0.32
Vitamin/trace mineral mix	10.0	10.0
DL-methionine	2.40	2.08
L-lysine-HCl	2.23	1.28
L-threonine	0.13	
Biofeed + CT	0.40	0.50
Nutrients (g/kg) (calculated)		
CP	211.1	209.2
AME_n, MJ/kg	12.42	12.85
$Isoleucine_{ad}$	7.7	7.5
$Leucine_{ad}$	13.6	12.9
$Lysine_{ad}$	10.8	9.9
$Meth._{ad}$ + $Cyst._{ad}$	7.9	7.5
$Phenyl._{ad}$ + $Tyr._{ad}$	14.1	13.7
$Threonine_{ad}$	6.5	6.2
$Tryptophan_{ad}$	2.0	2.0
$Valine_{ad}$	8.4	8.4
$Arginine_{ad}$	11.5	11.3
$Histidine_{ad}$	4.2	4.1
$NEAA_{ad}$	92.0	91.4

[1] containing 100 mg/kg monensin and 0.04 g/kg STAFAC (virginiamycine - 50 %)
[2] containing 1 mg/kg diclazuril and 0.04 g/kg STAFAC (virginiamycine - 50 %) (except for the last 5d)

Table 9 : Effect of early feed restriction, strain and sex on performance at 8 d of age (mean±SD)

	feed intake (g/d) (1-8 d)	body weight (g)	body weight gain (g/d) (1-8 d)	FC (1-8 d)
feed restriction				
ad libitum	18.2±1.4[a] [3]	146±13[a]	13.2±1.3[a]	1.386±0.053[a]
80 % - 4 d[4]	14.7±1.2[c]	133±11[c]	11.6±1.1[c]	1.263±0.055[c]
90 % - 4 d	16.0±1.3[b]	140±13[b]	12.4±1.3[b]	1.291±0.063[b]
80 % - 8 d	14.6±1.3[c]	130±9[d]	11.1±0.9[d]	1.317±0.057[ab]
SEM	0.12	1.09	0.14	0.014
Strain				
Ross	14.7±1.5[a]	127±7[a]	11.1±0.9[a]	1.322±0.084
Hybro	17.0±1.6[b]	148±9[b]	13.1±1.1[b]	1.307±0.059
SEM	0.09	0.77	0.10	0.010
Sex				
Male	16.0±2.1	138±15	12.1±1.6	1.322±0.068
Female	15.8±1.8	137±11	12.1±1.2	1.306±0.076
SEM	0.09	0.77	0.10	0.010
ANOVA (*p*-values)				
feed restriction (A)	0.000	0.000	0.000	0.000
strain (B)	0.000	0.000	0.000	0.266
sex (C)	0.200	0.575	0.963	0.260
B x C		0.006	0.012	
A x B				0.046
A x B x C				0.031

During the period after restriction, up to d 21, the same growth was achieved by the '90 % - 4 d'-treatment as in the *ad libitum* group (516 g), in spite of the significantly lower body weight at d 8. Birds restricted to 80 % of *ad libitum* intake for 4 or 8 d did not yet catch up with the control birds as growth rate and body weights at 21 d of age were significantly lower. FC was still slightly better for the restricted birds. However, there were no longer differences between treatments. No significant interactions were found.

The difference in body weight between the two strains remained at 21 d of age (Table 10). FC was similar for both strains and sexes. For the other performance variables, differences between sexes were as expected. Male broilers were heavier, had a higher feed intake and body weight gain during these first 3 weeks of the trial.

Table 11 summarises the performance at d 42 as influenced by feed restriction, strain and sex. A restriction to 90 % during a period of 4 d resulted in a final body weight not significantly different from but somewhat lower in comparison with the *ad libitum* group. The other

[3] means with the same letter are not significantly different from each other ($p<0.05$)

[4] 80 % - 4 d : restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 4 d
90 % - 4 d : restricted to 90 % of the *ad libitum* intake starting day 4 and lasting for 4 d
80 % - 8 d : restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 8 d

restrictions were too severe to catch up with the controls.

Table 10 : Effect of early feed restriction, strain and sex on performance at 21 d of age (mean±SD)

	feed intake (g/d) (1-21 d)	body weight (g)	body weight gain (g/d) (1-21 d)	FC (1-21 d)
feed restriction				
ad libitum	44.5±2.6^{a}[5]	662±41^{a}	29.5±1.9^{a}	1.507±0.025^{b}
80 % - 4 d[6]	41.7±1.7^{c}	626±28^{b}	27.9±1.3^{b}	1.494±0.017^{ab}
90 % - 4 d	43.6±2.3^{b}	656±33^{a}	29.3±1.4^{a}	1.487±0.020^{a}
80 % - 8 d	39.8±2.1^{d}	603±35^{c}	26.8±1.6^{c}	1.486±0.016^{a}
SEM	0.27	5.05	0.24	0.006
Strain				
Ross	41.0±2.3^{a}	615±32^{a}	27.5±1.5^{a}	1.493±0.024
Hybro	43.7±2.7^{b}	658±38^{b}	29.3±1.8^{b}	1.494±0.018
SEM	0.19	3.57	0.17	0.004
Sex				
Male	43.7±2.6^{a}	656±39^{a}	29.3±1.8^{a}	1.493±0.023
Female	41.0±2.3^{b}	617±34^{b}	27.5±1.6^{b}	1.494±0.019
SEM	0.19	3.57	0.17	0.004
ANOVA (*p*-values)				
feed restriction	0.000	0.000	0.000	0.056
Strain	0.000	0.000	0.000	0.814
Sex	0.000	0.000	0.000	0.943

Also, feed intake remained significantly lower for all restricted groups than in the *ad libitum* fed controls. The data in Table 11 show that FC was no longer significantly better in comparison with the *ad libitum* fed chickens. FC of Ross chickens was better than in Hybro, although the difference was not significant. As expected males converted their feed more efficiently than females (lower fat deposition for male broilers). At slaughter age, Hybro chickens were significantly heavier than Ross chickens.

[5] means with the same letter are not significantly different from each other ($p<0.05$)

[6] 80 % - 4 d: restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 4 d
90 % - 4 d: restricted to 90 % of the *ad libitum* intake starting day 4 and lasting for 4 d
80 % - 8 d: restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 8 d

Table 11 : Effect of early feed restriction, strain and sex on performance at 42 d of age (mean±SD)

	feed intake (g/d) (1-42 d)	body weight(g)	body weight gain (g/d) (1-42 d)	FC (1-42 d)
feed restriction				
ad libitum	89.1±6.9^{a}[7]	2186±190^{a}	51.1±4.5^{a}	1.747±0.038
80 % - 4 d[8]	86.6±5.8^{b}	2132±162^{bc}	49.8±3.8^{bc}	1.740±0.040
90 % - 4 d	87.5±6.2^{b}	2151±146^{ab}	50.2±3.4^{ab}	1.742±0.037
80 % - 8 d	84.7±6.2^{c}	2110±180^{c}	49.2±4.3^{c}	1.721±0.039
SEM	0.50	13.7	0.33	0.010
Strain				
Ross	84.3±5.9^{a}	2086±157^{a}	48.8±3.7^{a}	1.729±0.034
Hybro	89.7±5.5^{b}	2203±160^{b}	51.4±3.8^{b}	1.746±0.042
SEM	0.35	9.67	0.23	0.007
Sex				
Male	92.1±3.6^{a}	2291±85^{a}	53.6±2.0^{a}	1.720±0.038^{a}
Female	81.8±3.6^{b}	1999±74^{b}	46.6±1.7^{b}	1.755±0.031^{b}
SEM	0.35	9.67	0.23	0.007
ANOVA (*p*-values)				
feed restriction	0.000	0.003	0.003	0.268
Strain	0.000	0.000	0.000	0.088
Sex	0.000	0.000	0.000	0.001

3.2. Compensatory growth

Compensatory growth occurs when chickens whose growth was retarded by dietary restriction grow at a faster rate than animals of the same age that had no prior restriction. Using the Gompertz equation an estimation of the growth was made for both strains and sexes as a function of age for the different dietary restrictions and the *ad libitum* group (Figures 16-19). It seems that Ross males were not able to accomplish any compensatory growth (Figure 16). However, Ross female chickens restricted at 90 % for 4 d had already 'catch-up' growth at 3 weeks of age (Figure 17). This resulted in a final body weight similar to that of the control group, namely 1979 g (control: 1962 g). The most severe restriction (80 % - 8 d) caused an increased growth rate for Ross females but only during the last few days of the trial. This was not sufficient to catch up with the control group (1876 g and 1962 g respectively). Ross females showed no compensatory growth after being restricted to 80 % during 4 d. A final body weight of 1954 g was reached.

[7] means with the same letter are not significantly different from each other (p<0.05)
[8] 80 % - 4 d: restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 4 d
90 % - 4 d: restricted to 90 % of the *ad libitum* intake starting day 4 and lasting for 4 d
80 % - 8 d: restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 8 d

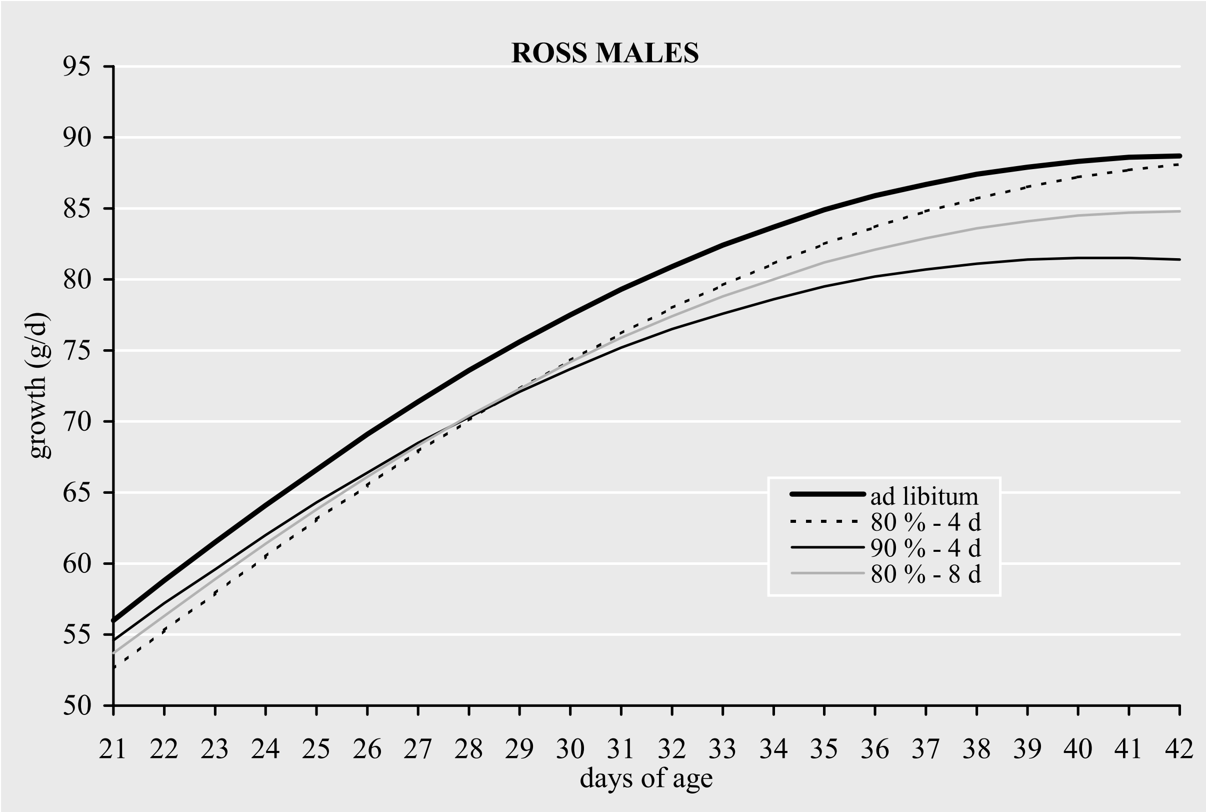

Figure 16 : Effect of feed restrictions on growth for Ross male broilers between 21 and 42 d of age

ad libitum: $W_t = 38.4\ e^{(0.1979/0.0389)(1-e^{-0.0389t})}$ 80 % - 4 d : $W_t = 38.4\ e^{(0.1906/0.0372)(1-e^{-0.0372t})}$

90 % - 4 d : $W_t = 38.4\ e^{(0.1996/0.0402)(1-e^{-0.0402t})}$ 80 % - 8 d : $W_t = 38.4\ e^{(0.1904/0.0372)(1-e^{-0.0372t})}$

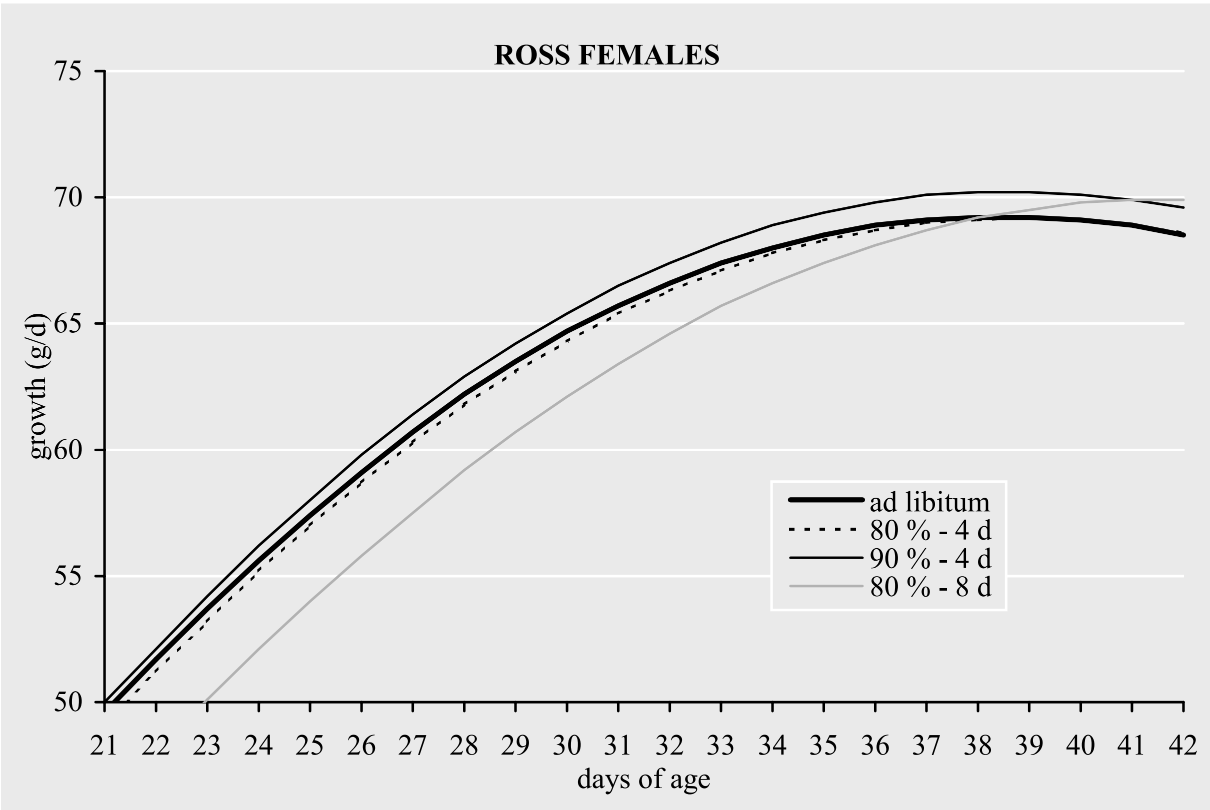

Figure 17 : Effect of feed restrictions on growth for Ross female broilers between 21 and 42 d of age

ad libitum: $W_t = 37.9\ e^{(0.1978/0.0413)(1-e^{-0.0413t})}$ 80 % - 4 d : $W_t = 37.9\ e^{(0.1965/0.0409)(1-e^{-0.0409t})}$

90 % - 4 d : $W_t = 37.9\ e^{(0.1981/0.0416)(1-e^{-0.0412t})}$ 80 % - 8 d : $W_t = 37.9\ e^{(0.1871/0.0384)(1-e^{-0.0384t})}$

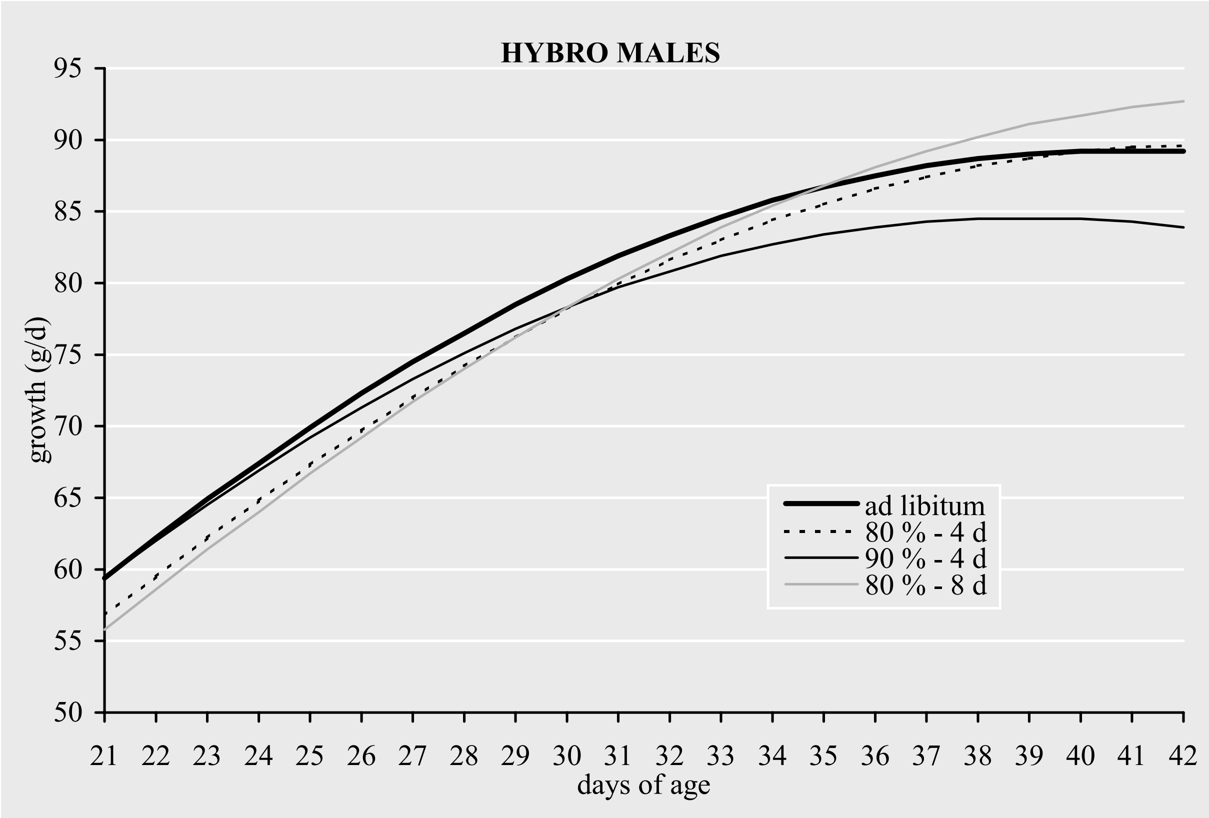

Figure 18 : Effect of feed restrictions on growth for Hybro male broilers between 21 and 42 d of age

ad libitum: $W_t = 43.9\ e^{(0.1957/0.0396)(1-e^{-0.0396t})}$ 80 % - 4 d : $W_t = 43.9\ e^{(0.1898/0.0381)(1-e^{-0.0381t})}$

90 % - 4 d : $W_t = 43.9\ e^{(0.1991/0.0411)(1-e^{-0.0411t})}$ 80 % - 8 d : $W_t = 43.9\ e^{(0.1858/0.0368)(1-e^{-0.0368t})}$

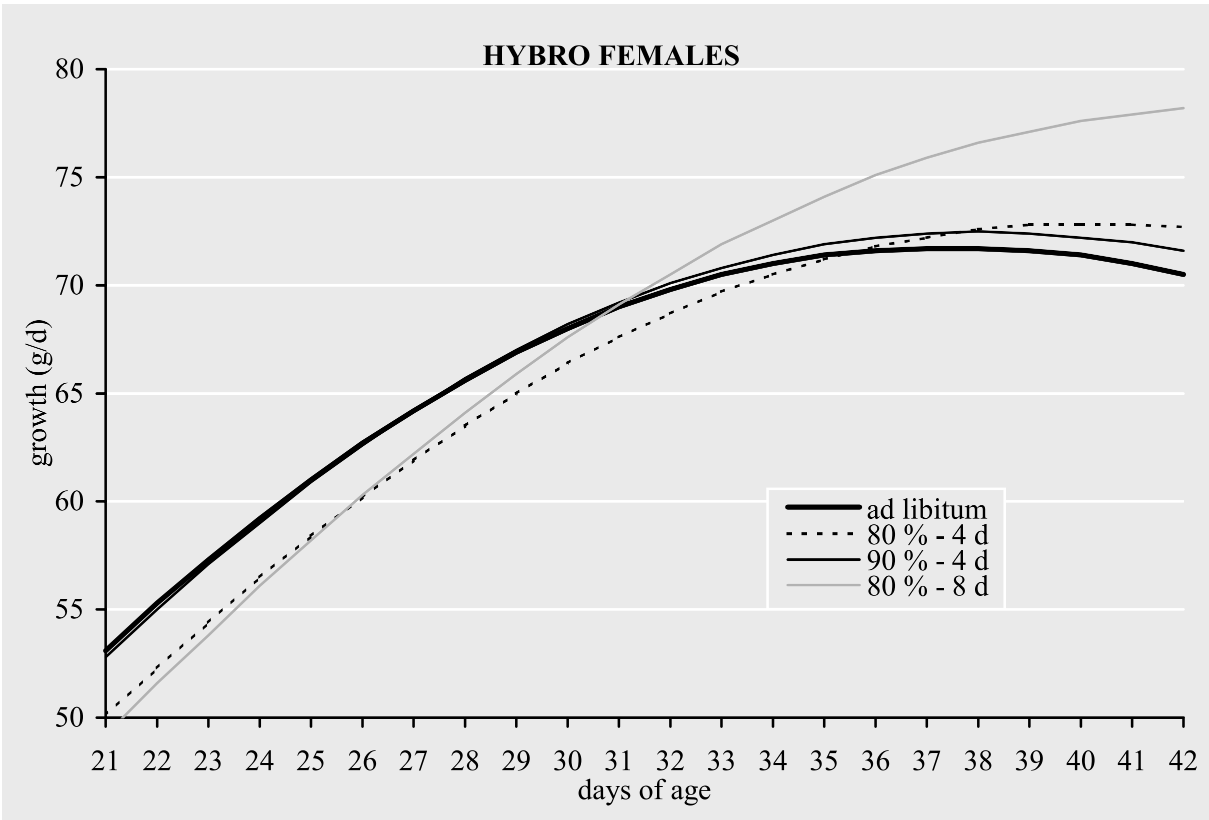

Figure 19 : Effect of feed restrictions on growth for Hybro female broilers between 21 and 42 d of age

ad libitum: $W_t = 43.1\ e^{(0.1959/0.0418)(1-e^{-0.0418t})}$ 80 % - 4 d : $W_t = 43.1\ e^{(0.1871/0.0393)(1-e^{-0.0393t})}$

90 % - 4 d : $W_t = 43.1\ e^{(0.1943/0.0413)(1-e^{-0.0413t})}$ 80 % - 8 d : $W_t = 43.1\ e^{(0.1809/0.0370)(1-e^{-0.0370t})}$

There was little compensatory growth for Hybro males (Figure 18). The restriction 90 % - 4 d caused only a brief period of increased growth shortly after the restriction period. After which growth rate dropped below that of the controls to stay rather constant during the last week. Hybro males being restricted to 80 % during 8 d grew faster than the controls only during the last days of the trial. In all of these cases Hybro males had a final body weight about 70 g lower than the *ad libitum* group (±2330 g and ±2400 g, respectively). These differences were not significant. Restricting Hybro females for 4 d to 90 % of the *ad libitum* intake resulted in a compensatory growth during the last 10 d (Figure 19). Final body weight of this group did not differ from the controls (2079 g and 2077 g, respectively). The severe restriction (80 % - 8 d) also caused 'catch-up' growth during the last week, which was much more pronounced even though mean final body weight only reached 2035 g (not significantly different from control weight of 2077 g). When this restriction lasted only 4 d, compensatory growth was delayed until the last days of the trial. No complete 'catch-up' to the final body weight of the controls was accomplished (2025 g).

3.3. Mortality and uniformity

Table 12 shows a slight trend towards a reduced total mortality (dead + removed) with feed restrictions, except for treatment 80 % - 8 d. Also death by SDS tended to be lower when chickens are retarded in early growth. Uniformity at 42 d of age was significantly reduced (3.7 %) when the chickens were restricted to 80 % during 8 d (Table 12). The milder feed restrictions had no significant effect on uniformity.

Table 12 : Effect of early feed restriction on mortality[9] and uniformity (mean±SD)

	total mortality (%)[10]	SDS (%)	removed with leg problems (%)	uniformity
ad libitum	5.4±4.3	0.9±1.1	0.1±0.3[ab11]	94.6±2.6[a]
80 % - 4 d[12]	4.9±2.9	0.7±0.9	0.6±0.9[b]	92.0±3.2[ab]
90 % - 4 d	3.8±5.7	0.3±0.8	0.0±0.0[a]	93.9±4.8[ab]
80 % - 8 d	6.1±4.4	0.6±0.9	0.7±1.0[b]	90.9±5.4[b]

[9] no cases of ascites were detected
[10] significantly more chickens were removed within the Ross 508 line in comparison with the Hybro chickens
[11] means with the same letter are not significantly different from each other ($p<0.05$)
[12] 80 % - 4 d : restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 4 d
90 % - 4 d : restricted to 90 % of the *ad libitum* intake starting day 4 and lasting for 4 d
80 % - 8 d : restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 8 d

3.4. Carcase composition

Protein and lipid content (g/kg total animal, feathers inclusive) were not changed significantly by feed restriction (Table 13). However a significant interaction between restriction and sex in total protein content was found (Figure 20). Indeed, restricting male chickens to 80 % of *ad libitum* for 8 d increased the protein content whereas the opposite seemed to be true for female chickens. At the age of 42 d the abdominal fat content was increased for birds restricted during early life. No significant difference in abdominal fat was observed between the different restrictions. In contrast, the restriction programmes did not affect total body lipid content.

The results show that Ross chickens had a higher protein content and were leaner. Expected differences between males and females were confirmed. Interactions between sex and strain for total lipid content and abdominal fat content were significant. The lipid content of Ross males was only 2.1 % less than that of Ross females. For the Hybro chickens the difference between the two sexes was 3.5 %. A similar trend was observed for the abdominal fat content. The difference for Ross chickens was 0.52 % and 0.83 % for the Hybro strain.

The results of feed restriction, strain and sex on cut-up parts are summarised in Table 13. Feed restriction caused a reduction in carcase yield, which was significant for the 80 % - 8 d treatment. Breast meat proportion, however, was not significantly affected by early feed restriction. None of the other carcase parts were significantly influenced by the restrictions, although there was a significant interaction between restriction and sex for the proportion of wings (Figure 21). Indeed, restricting male broilers to 90 % - 4 d decreased the percentage wings whereas the opposite occurred for the female chickens.

Ross birds had a significantly higher carcase yield and breast meat percentage than Hybro, which was compensated for by a reduction of the other carcase parts. Breast meat percentage was not different between sexes. Drumstick percentage was significantly higher for male than for female chickens, with the difference between the two sexes more pronounced for the Hybro than for the Ross chickens (strain x sex interaction). Thigh percentage was lower for the males. The percentage of wings was lower with the males of the Ross strain than with the females of the Ross strain; the opposite was true for the Hybro chickens (strain x sex interaction).

Table 13 : Influence of feed restriction, strain and sex on protein and lipid content (g/kg whole bird inclusive feathers), abdominal fat content, carcase yield and cut-up parts (42 days of age) (mean±SD)

	protein content	lipid content	abd. fat (g/kg)	yield (%)	breast meat (%)	drumstick (%)	thigh (%)	wings (%)	bones + skin (%)
feed restriction									
ad libitum	184.5±3.7^{ab}[13]	145.2±16.9	20.8±3.3^{a}	68.3±0.7^{bc}	26.5±1.3	14.1±0.6	30.0±0.5^{a}	11.8±0.3	17.7±0.7
80% - 4 d[14]	184.2±5.3^{ab}	146.6±14.5	22.3±4.4^{b}	68.0±0.7^{ab}	25.9±1.4	14.1±0.7	30.1±0.6^{ab}	11.8±0.3	18.1±0.7
90% - 4 d	181.5±5.1^{a}	152.0±19.6	22.3±4.9^{ab}	68.5±0.9^{c}	26.1±1.4	14.1±0.7	30.0±0.8^{a}	11.8±0.3	18.0±0.7
80% - 8 d	187.1±11.5^{b}	149.4±19.3	22.0±4.2^{ab}	67.8±0.5^{a}	26.1±1.2	14.0±0.6	30.4±0.8^{b}	11.8±0.3	17.7±0.6
SEM	1.52	2.68	0.53	0.16	0.20	0.09	0.15	0.07	0.19
strain									
Ross	187.1±7.8^{a}	144.2±13.7^{a}	20.5±3.1^{a}	68.6±0.6^{b}	27.2±0.6^{b}	13.8±0.4^{a}	29.7±0.4^{a}	11.6±0.2^{a}	17.7±0.5
Hybro	181.5±5.2^{b}	152.4±19.8^{b}	23.2±4.7^{b}	67.7±0.6^{a}	25.1±0.8^{a}	14.4±0.6^{b}	30.6±0.7^{b}	11.9±0.3^{b}	18.0±0.8
SEM	1.08	1.89	0.38	0.11	0.14	0.06	0.11	0.05	0.13
sex									
male	186.1±8.7^{a}	134.4±7.5^{a}	18.5±1.5^{a}	68.1±0.8	26.0±1.4	14.5±0.6^{a}	30.0±0.7^{a}	11.8±0.3	17.7±0.7
female	182.6±4.7^{b}	162.2±12.3^{b}	25.2±3.1^{b}	68.1±0.7	26.3±1.2	13.7±0.3^{b}	30.3±0.7^{b}	11.8±0.2	18.0±0.6
SEM	1.08	1.89	0.38	0.11	0.14	0.06	0.11	0.05	0.13
ANOVA (*p*-values)									
feed (A)	0.105	0.308	0.161	0.016	0.283	0.961	0.110	0.999	0.327
strain (B)	0.001	0.005	0.000	0.000	0.000	0.000	0.000	0.000	0.077
sex (C)	0.027	0.000	0.000	0.861	0.286	0.000	0.017	0.566	0.122
B x C		0.014	0.006			0.010		0.025	
A x C	0.002							0.017	

[13] means with the same letter are not significantly different from each other ($p<0.05$)

[14] 80 % - 4 d : restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 4 d
90 % - 4 d : restricted to 90 % of the *ad libitum* intake starting day 4 and lasting for 4 d
80 % - 8 d : restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 8 d

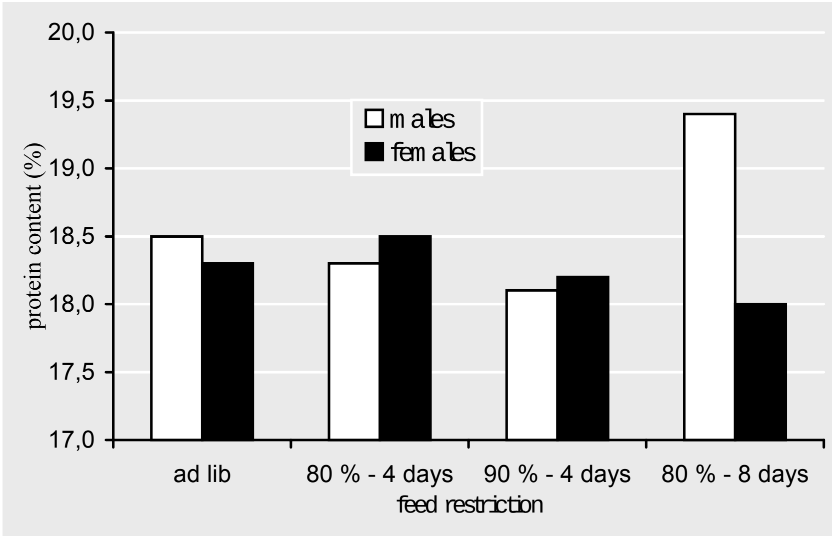

Figure 20 : Difference between male and female chickens in effect of feed restriction on protein content (g/kg whole animal inclusive feathers) at the age of 42 d

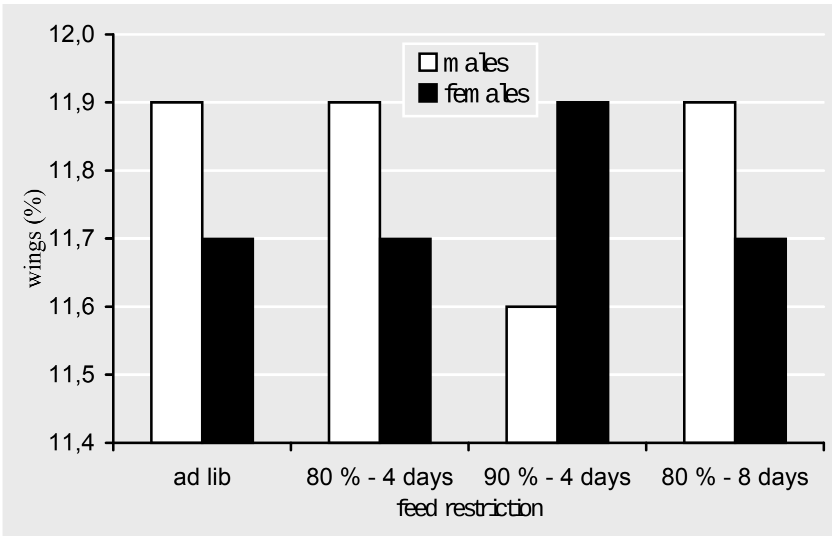

Figure 21 : Difference between male and female chickens in effect of feed restriction on the percentage of wings at the age of 42 d

3.5. Meat quality

Normally, muscle pH is about 7 and rapidly declines after slaughter. In this trial the meat pH, 24 h after slaughter, reached an average of 5.73. Treatment, strain or sex had no significant effect on this variable (Table 14).

Only sex had a significant effect on meat colour. The L-value was higher for female then for male. The b-parameter was also significantly higher for females. So, meat from female birds seems to be paler than that from males. The meat from Ross birds showed, on average, a lower loss of moisture under pressure (higher water holding capacity) than Hybro, corresponding with the lower cooking and drip losses.

Shear force as a measure for tenderness of chicken meat was higher for male birds (not significant). No significant difference was noticed between Ross and Hybro. For the 90 % - 4 d treatment a lower shear force in comparison with the other treatments was measured.

4. Discussion

The present study indicates that chickens subjected to an early feed restriction of about 90 % of the *ad libitum* intake during 4 d have a comparable final body weight to that of the control group. A lower body weight was obtained with the other, more severe, restrictions. Weights at 21 days of age for the 90 %-group and 80%-group, respectively, were 1 % and 5.4 % lower in comparison with the control group. These growth retardations are very low in comparison with the recommendations mentioned in the manual guide of Ross Breeders (1999), being 8-12 % of the control group. When considering the growth retardations by strain and by sex, no correlation is found between 21 d liveweight reduction and the differences at 42 days (results not shown). A trend of improvement in feed conversion was noted only with the highest restriction level (80 %). These results agree with those reported by Deaton (1995), who found that a restriction of 90 % (from d 7 to d 14) resulted in a complete recovery of the body weight at the age of 41 d without improvement in feed conversion. When restrictions of 75 or 60 % were used during the same period, a significant improvement in feed conversion can be accomplished. Like in the studies of Mollison *et al.* (1984), Pinchasov and Jensen (1989), Cristofori *et al.* (1997), the improved feed conversion was at the expense of final body weight. On the other hand, Plavnik and Hurwitz (1985, 1988 and 1991) could achieve a better feed conversion without reduction of the body weight at slaughter.

Table 14 : Effect of early feed restriction, strain and sex on meat quality (mean±SD)

	pH_{24}	colour			moisture (%)	drip (%)	cooking losses (%)	shear force (N)
		L	a	b				
feed restriction								
ad libitum	5.70±0.07	56.6±1.6	7.15±0.96	14.8±1.0[b][15]	24.2±5.6	6.9±1.5	15.0±1.2	21.4±8.4[ab]
80 % - 4 d[16]	5.73±0.07	56.6±2.3	6.98±1.29	14.6±1.1[ab]	22.1±4.2	6.5±1.4	15.0±1.3	24.1±9.1[b]
90 % - 4 d	5.75±0.11	57.0±2.0	6.64±1.39	14.4±1.4[ab]	22.1±4.2	5.7±1.9	14.2±1.2	18.6±5.9[a]
80 % - 8 d	5.77±0.08	55.5±1.8	7.58±1.41	13.9±0.9[a]	22.8±4.3	6.9±3.1	15.0±2.1	21.7±6.8[ab]
SEM	0.02	0.55	0.39	0.27	0.13	0.59	0.47	176
strain								
Ross	5.75±0.09	56.5±2.0	6.86±1.24	14.3±0.9	21.4±3.6[a]	6.0±1.8	14.6±1.6	21.0±6.3
Hybro	5.71±0.07	56.3±1.9	7.32±1.31	14.5±1.2	24.2±5.0[b]	7.0±2.2	15.0±1.4	21.9±9.3
SEM	0.02	0.39	0.28	0.19	0.09	0.42	0.34	1.24
sex								
male	5.74±0.10	55.7±2.0[a]	7.32±1.46	13.9±1.0[a]	22.6±4.1	6.5±2.2	14.7±1.6	22.1±8.1
female	5.73±0.07	57.1±1.7[b]	6.85±1.05	14.9±1.0[b]	23.0±5.1	6.6±1.9	14.9±1.4	20.7±7.6
SEM	0.02	0.39	0.28	0.19	0.09	0.42	0.34	1.24
ANOVA (*p*-values)								
feed restriction	0.244	0.261	0.417	0.191	0.635	0.463	0.590	0.204
strain	0.110	0.626	0.249	0.593	0.041	0.113	0.443	0.643
sex	0.527	0.012	0.244	0.001	0.790	0.882	0.676	0.446

[15] means with the same letter are not significantly different from each other ($p<0.05$)

[16] 80 % - 4 d : restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 4 d
90 % - 4 d : restricted to 90 % of the *ad libitum* intake starting day 4 and lasting for 4 d
80 % - 8 d : restricted to 80 % of the *ad libitum* intake starting day 4 and lasting for 8 d

The variability of results found in the literature is due to a number of factors that influence the responses of broilers to early feed restriction. These factors include the nature, timing, severity and duration of the feed restriction but also genetic factors, such as strain and sex. The present results show that, if realised, the initiation and the magnitude of the established compensatory growth are different between the Ross and Hybro strains and between males and females. Generally it is stated that male broilers have a greater ability to exhibit compensatory growth following a period of undernutrition than females (Plavnik and Hurwitz, 1988; Plavnik and Hurwitz, 1991). The opposite seemed to be true in this study. The differences between sexes in the Hybro chickens were similar but less pronounced than in Ross chickens. On average, the magnitude of the 'catch-up' growth established by the Hybro chickens was higher than in the Ross chickens. This may be explained by the fact that the Ross 508 strain is already genetically selected for lower juvenile growth followed by compensatory growth. For this reason, possibilities for compensatory growth induced by an 'extra' (feed) restriction of early growth might be limited. In addition, the average initial body weight, 38.1 g, of the Ross chickens was rather low. Feed restriction, combined with these low weights, might be another limitation for compensatory growth. Indeed other results obtained in our Institute showed good compensatory growth for Ross chickens (508) with higher initial hatching body weights (unpublished data).

In general, compensatory growth often did not occur until the last days of the trial or did not occur at all within this period. This is in contrast with the statement of Wilson and Osbourn (1960, cited by Zubair and Leeson, 1996a) that compensatory growth after a period of undernutrition is greatest immediately following realimentation. Leeson and Zubair (1997) found 'catch-up' growth between d 12 and d 21 following feed restriction between d 6 and d 12. Saleh *et al.* (1996) found a compensatory growth between d 15 and d 21 after a discontinuous restriction of 30 % of *ad libitum* during the period of 8 to 13 d of age (day 10 and 11 *ad libitum*). A discontinuous restriction of 20 % or 40 % of the *ad libitum* intake during that same period only gave compensatory growth within the period of 22 to 35 d of age.

An important factor influencing the variability observed in compensatory growth might be the conditions of realimentation. There is at present very limited information about nutrient requirements during the refeeding period. The plateau in the growth noticed during the last week with both Ross and Hybro birds restricted to 90 % might be an indication of the need for higher levels of energy and/or amino acids during the period of refeeding. More research is needed to get clear information about these requirements.

There was a tendency for reduced total mortality and death due to SDS. Because the data were from a very small number of animals, the effect of restriction on mortality and leg problems may have been obscured. Indeed, Scheideler and Baughman (1993) also found no significant effect of feed restriction on the number of dead birds in a trial with a normal mortality rate, but early feed restriction decreased mortality due to heat stress substantially but not significantly in a second trial. Results from Van Harn and Fabri (1995) with a larger number of chickens (2 x 12,600) however, showed that a feeding programme in combination with a lighting schedule can reduce total mortality from 6.8 % to 3.7 %.

One of the most controversial aspects of early feed restriction programmes has been the lack of a consistent effect on abdominal fat pad or total carcase lipid. A reduction in body fat and/or abdominal fat content without any concomitant reduction in body weight was found by Plavnik and Hurwitz (1985, 1988, 1991) and Jones and Farrell (1992b). Others have reported reductions in abdominal fat pad or total carcase fat due to feed restriction early in life but with a small reduction in final body weight (Mollison *et al.*, 1984; Cabel and Waldroup, 1990; Mudrić *et al.*, 1994). Others (Pinchasov and Jensen, 1989; Leeson *et al.*, 1991; Fontana *et al.*, 1993; Scheideler and Baughman, 1993; Susbilla *et al.*, 1994; Deaton, 1995; Palo *et al.*, 1995; Cristofori *et al.*, 1997) were not able to show a clear effect. Beane *et al.* (1979) reported that feed efficiency was improved, but the amount of abdominal fat in male broilers was significantly increased. This agrees with the present results, although differences were not significant. Total lipid content however, was not changed by the feed restrictions. Moreover, it seems that the increase in the abdominal fat percentage resulting from restrictions was more pronounced with the female broilers than with the males (not significant). It can not be excluded that differently located fat depots are differentially affected by the altered growth trajectory induced by a temporary growth retardation at young age. Indeed, differences in the maturing rate (cf. the allometric coefficients) of the different fat depots are found comparing birds reared in a continuous lighting programme or an intermittent lighting programme (Buyse, 2001).

In contrast with the situation with carcase or abdominal fat content, few results on effects of feed restriction on slaughter yield, cut-up parts and meat quality are mentioned in the literature. Scheideler and Baughman (1993) found a reduction in percentage of dressed carcase yield of 42-d old restricted broilers in one trial but not in a second. Also Leeson *et al.* (1991), Van Harn (1992), Zubair and Leeson (1994a) and Palo *et al.* (1995) could not demonstrate any effect of feed restriction on dressing percentage. In the present trial a reduction was found only with the most severe restriction. However, data of Saleh *et al.* (1996) showed a trend of increasing dressing percentage for the restricted birds as well as an improvement in breast meat yield. No effect was found on the latter parameter by Pinchasov and Jensen (1989), Leeson *et al.* (1991), Van Harn (1992), Carter *et al.* (1994), Zubair and Leeson (1994a), Palo *et al.* (1995) and this was confirmed by our own results. On the other hand, Van Harn and Fabri (1995) found a negative effect of using a feeding programme (in combination with a lighting schedule) on proportion of breast meat. The percentage of leg meat in their trial, however, tended to be higher with restriction. Other researchers found no significant effect of feed restriction on the percentage of thighs or drumsticks (Van Harn, 1992; Susbilla *et al.*, 1994; Saleh *et al.* 1996) and this was confirmed in our study.

Also little information is available about the effects of feed restriction on meat quality. Smoliñska *et al.* (1994) concluded that meat colour is significantly affected by nutrition and sex. Indeed, in our trial there was only a difference between males and females. The fact that female broilers seemed to be paler might be related to the higher lipid content of the carcases. No effect of feed restriction on meat colour was noticed. Other results obtained in our Institute confirm this (unpublished data).

Close (1997) demonstrated that *ad libitum* fed pigs produced more tender meat than pigs fed to only 80 % of *ad libitum* intake. He concluded that a faster growth and, by implication, *ad libitum* feeding may produce more tender pig meat. This is in contrast with the findings of Schreurs (1998) who found that fast growing chicken strains produced less tender meat in comparison with slow growing strains. However, the fact that genetically very different strains were used might be of more importance than the difference in growth rate itself. In the present trial, no significant effect of feed restriction on meat tenderness was found.

In conclusion, the evidence suggests that using feed restriction doesn't always give an answer to all of the problems associated with fast growing broilers. However, an early mild restriction (90 % during 4 d) gave noteworthy results. It induced a compensatory growth sufficient to reach a normal final body weight without a negative effect on feed conversion, flock uniformity or carcase and meat quality. Female broilers even reached a somewhat higher body weight than the *ad libitum* group. Along with these observations, there seems to be a possibility of reducing total mortality and the occurrence of SDS. Although total lipid content was not changed, the abdominal fat percentage increased (not significant). More research is needed to fully understand the mechanism of compensatory growth, protein turnover and specific nutrient requirements during this period.

Chapter 5

QUALITATIVE FEED RESTRICTION OF BROILER CHICKENS

Adapted from :

Lippens, M., Huyghebaert, G., Van Tuyl, O. & De Groote, G. (2002). Early and temporary qualitative, autonomous feed restriction of broiler chickens. Effects on performance characteristics, mortality, carcass and meat quality. Archiv für Geflügelkunde 67(2) : 49-56.

ABSTRACT

The objective of the present trial was to examine the effect of a qualitative feed restriction during the early stage of life on performance characteristics, mortality, carcase quality and meat quality of two modern broiler strains. It was chosen to use a low energy diet or a NaCl-deficient diet offered from the 4th day during 4 days. A 3-factorial experiment (3 x 2 x 2) was set up with the two strains, Ross 508 and Hybro G. Sexes were separated.

Results show that the qualitative feed restrictions used were effective ways to induce a compensatory growth after the temporary growth retardation. Indeed, final body weights of the chickens subjected to these restrictions showed no significant differences with the control birds. On the other hand, due to the rather low number of animals used, no significant reduction of losses due to metabolic diseases were found. Only a positive trend on mortality due to SDS could be found. However, the economical impact in practice of these figures can still be important.

There was no indication in the present trial that changes in growth pattern (growth retardation - compensatory growth) induced by qualitative feed restriction had a negative effect on slaughter yield or cut-up parts or are of that nature that meat quality is impaired.

In conclusion, qualitative feed restrictions as used in the present trial, gave some indications of being a practical tool to reduce losses due to metabolic diseases without deteriorating performance or carcase quality. More research however is needed to confirm this.

1. Introduction

Modern broiler strains are characterised by a very high growth rate and a low feed conversion ratio. On the other hand, high incidences of metabolic diseases, leg problems and an increased fat deposition are typical for these extreme selected lines. These negative aspects are of major concern for the farmer and processor, because they can bring about important economic losses. Earlier research indicated that a mild quantitative feed restriction during early life may offer some possibilities to limit these important economic losses mentioned, without any deterioration of performance (Plavnik and Hurwitz, 1985; 1988; 1991; Deaton, 1995; Lippens *et al.*, 2000; Lippens *et al.*, 2002a). The application of these quantitative techniques in practice sometimes may cause, practical problems. Therefore, the method of intake restriction of broiler chickens by qualitative means was tested in the present trial.

Although in the past a great deal of research has been done on these kind of restrictions for rearing broiler breeder pullets (for reviews see Lee *et al.*, 1971; Van Wambeke, 1977, 1981, 1989; De Groote, 1996), studies concerning broilers however are limited. Moreover, the effect of such a feed restriction on meat quality is still missing in literature.

As nowadays the consumer puts high demands upon animal welfare, production systems and the use of artificial feed additives, two qualitative feed restrictions were chosen taking in account these aspects. Dilution of the diet with oat hulls, rice bran, cellulose or another inert filler can be a rather easy way to induce growth retardation. Leeson *et al.* (1991) found a complete recovery of body weight at the age of 42 d, after dilution of the diet with ground rice hulls up to 55 % during the period 4 to 11 d of age. The overall feed efficiency was not affected. However, there was an indication of reduced abdominal fat content for males at 56 days. Similar results were obtained by Jones and Farrell (1992a) and Zubair and Leeson (1994a) who fed diets diluted up to 65 % with rice hulls and up to 50 % with oat hulls, respectively.

In contrast, own research in our Institute indicated that when lowering both energy and protein content of a wheat based diet with 30 % during the restricted period (for 7 d starting from d 4), chickens were unable to realise the same final body weights at 42 d of age compared with the controls (Lippens *et al.*, 2002a). Alternatively to a less severe restriction, lowering the energy content of the diet might be a way to give the chickens the possibility to establish the desired compensatory growth. Indeed, they can adjust themselves much better when only energy is reduced (Leeson *et al.*, 1996; Lippens *et al.*, 2002a).

A sodium-deficient diet also is known to reduce the appetite of the birds and can be used as an alternative to diet dilution. Using such a diet for 6 d with male broilers improved FC and reduced abdominal fat content at the age of 56 d in a trial of Plavnik and Hurwitz (1990). Feeding a low sodium diet for 9 d to female broilers induced a growth retardation which could not be recovered by the age of 56 d (Plavnik and Hurwitz, 1990). Ross female broilers restricted with a low sodium diet for 7 d, however, were able to catch up with the controls at the age of 42 d in a trial of Meluzzi *et al.* (1995). Abdominal fat content (as % of live weight), however, was not significantly reduced.
The objective of the present trial was to examine the effect of a low energy diet or a NaCl-deficient diet offered in the early stage of life on performance characteristics, mortality, carcase quality and meat quality of two modern broiler strains.

2. Materials and methods

2.1. Experimental design

A 3-factorial experiment (3 x 2 x 2) was set up with two commercial strains (Ross 508 and Hybro G) and both sexes. The experiment was conducted with 1398 Ross 508 and 1398 Hybro G day-old broiler chickens. The qualitative feed restrictions consisted of a low energy or NaCl-deficient diet. Each dietary treatment, per strain and per sex, had 3 replicates consisting of 2 large pens containing 100 birds and 1 small pen containing 33 birds.

2.2. Diets

Except for the duration of the qualitative restriction, a starter diet with 225 g CP/kg and 12.42 MJ AME_n (broilers; CVB, 1997)/kg was given until 14 d of age. From d 15 until d 42 a grower diet with 213 g CP/kg and 12.85 MJ AME_n/kg was offered. Next to the control, two different kinds of qualitative restrictions were used in the early stage of life. The first one consisted of feeding a low energy, normal protein diet (9.9 MJ AME_n/kg; 225 g CP/kg) from d 4 for 4 d. In a second treatment conducted for the same period, a diet with low NaCl-content (calculated concentration of 0.05 %) was offered. For the ingredient and chemical composition of the diets see Table 15.

Table 15 : Ingredient composition and calculated nutrient composition of the diets (g/kg, unless otherwise stated)

Ingredients	starter (0-14 d)[1]	low enery[1]	low NaCl[1]	grower (15-42 d)[2]
Wheat	332.4	463.9	340.6	500.0
Soybeans (full fat)	44.6	56.1	32.7	135.8
Soybean meal (44 % CP)	269.1	206.2	276.4	
Soybean meal (48 % CP)				117.5
Yellow corn	200.0		200.0	100.0
Oat hulls		134.9		
Animal fat	70.0	20.0	70.0	54.7
Meat meal (58 % CP, 14 % CF)	50.0	80.0	50.0	60.0
Dicalc. phosph.	13.77	9.54	13.7	7.88
Limestone	3.03	11.52	3.07	5.82
Sodium chloride	2.72	2.58		2.61
Sodium bicarbonate	0.79			0.74
Vitamin/trace mineral mix	10.0	10.0	10.0	10.0
DL-methionine	2.08	2.50	2.07	2.00
L-lysine-HCl	0.89	1.39	0.92	2.17
L-threonine	0.22	0.63	0.23	0.72
Biofeed+ CT	0.33	0.46	0.34	0.50
Sodium glutamate		0.21		
Nutrients (g/kg) (calculated)				
CP	225.0	225.0	225.0	213.0
AME_n, MJ/kg	12.42	9.90	12.42	12.85
$Isoleucine_{ad}$	8.4	7.9	8.4	7.6
$Leucine_{ad}$	14.7	13.0	14.7	13.1
$Lysine_{ad}$	10.8	10.8	10.8	10.7
$Meth._{ad}$ + $Cyst._{ad}$	7.9	8.0	7.9	7.5
$Phenyl._{ad}$ + $Tyr._{ad}$	15.3	14.1	15.2	13.9
$Threonine_{ad}$	7.2	7.2	7.2	7.0
$Tryptophan_{ad}$	2.2	2.1	2.2	2.0
$Valine_{ad}$	9.1	8.7	9.1	8.5
$Arginine_{ad}$	12.7	12.3	12.7	11.5
$Histidine_{ad}$	4.6	4.3	4.6	4.2
$NEAA_{ad}$	98.0	95.5	98.1	92.7
Ca	9.0	13.0	9.0	9.0
P	7.5	7.3	7.5	6.5
K	9.1	8.7	9.1	7.9
Na	1.8	1.8	0.5	1.8
Cl	2.3	2.5	0.7	2.5
dEB[3]	247	231	235	210

2.3. Response parameters

The general response parameters are mentioned in Chapter 3. At 43 d also, protein and lipid contents were measured on 9 chickens per pen (3 for the small pens).

[1] containing 100 mg/kg monensin and 0.04 g/kg STAFAC (virginiamycine - 50 %)
[2] containing 1 mg/kg diclazuril and 0.04 g/kg STAFAC (virginiamycine - 50 %) (except for the last 5 d)
[3] dietary electrolyte balance (K+Na-Cl)

3. Results

3.1. Performance

Both qualitative feed restrictions significantly reduced body weight at 8 d of age (Table 16). The low energy diet induced a reduction in the growth of 6.7 %, while the 'NaCl-treatment' lowered it with 11 %. The low energy diet however induced the strongest reduction in feed intake (8.7 % against 4.1 %). Feed restriction affected feed conversion ratios significantly with a significant deterioration of the FC for the 'low NaCl'-treatment only.
At the age of 8 d the body weights of the two strains were not significant different. FC was better for the Ross chickens. Male chickens were heavier than females. There was no difference in FC for the two sexes.
At the age of 21 d restricted birds were still smaller than the controls (Table 16). However, birds restricted with the low NaCl diet reached practically the same growth rate between d 8 and d 21 as the control birds (38.7 g/d and 38.8 g/d respectively) despite of their significant lower weights at d 8. The chickens restricted with the low energy diet reached an average growth of only 37.2 g/d for the same period. Compensatory growth seems to be more delayed for this treatment.
At this stage of age, the significant differences in FC due to restrictions had already disappeared. Ross birds however were on average more efficient in feed conversion than Hybro chickens. No sex-effect on feed conversion was found as the faster growth of the males is accompanied with a proportionally higher feed intake.
Table 17 shows the final performance results at 42 d of age. Compensatory growth was complete for both qualitative feed restrictions. The restriction with the 'low NaCl'-diet induced the highest 'catch-up'-growth. Indeed, these chickens had the lowest weights short after the restriction and the final body weight was the highest at finish, in absolute figures even higher than the control birds (not significantly different). The deterioration of the FC short after restrictions was reversed although differences were insufficient to be of any economic significance.
No significant strain effect on the performances at the age of 42 d was noticed, although Ross birds had a near to significant better feed conversion. Differences between the sexes were again as expected.

Table 16 : Effect of qualitative feed restriction, strain and sex on the performance at 8 d and 21 days of age (mean±SD)

	feed intake (1-8d) (g/d)	body weight d 8 (g)	FC (1-8d)	feed intake (1-21d) (g/d)	body weight d 21 (g)	FC (1-21d)
feed restriction						
ad libitum	19.5±0.9[c4]	163±4[c]	1.277±0.050[a]	45.9±1.7[b]	668±22[b]	1.539±0.036
low energy[5]	17.8±1.3[a]	152±6[b]	1.281±0.058[a]	43.8±1.9[a]	636±26[a]	1.548±0.037
NaCl	18.7±0.9[b]	145±3[a]	1.435±0.063[b]	44.4±1.7[a]	648±26[a]	1.537±0.038
strain						
Ross	18.0±0.9[a]	154±8	1.283±0.075[a]	44.1±1.7[a]	650±23	1.524±0.025[a]
Hybro	19.3±1.2[b]	152±9	1.379±0.087[b]	45.3±2.0[b]	651±32	1.559±0.038[b]
sex						
male	19.0±1.0[a]	156±9[a]	1.327±0.094	46.1±1.3[a]	668±18[a]	1.542±0.035
female	18.3±1.3[b]	151±9[b]	1.335±0.095	43.4±1.5[b]	633±24[b]	1.540±0.038
ANOVA (*p*-values)						
feed restriction	0.000	0.000	0.000	0.000	0.000	0.675
strain	0.000	0.182	0.000	0.001	0.822	0.004
sex	0.006	0.000	0.459	0.000	0.000	0.859

Table 17 : Effect of qualitative feed restriction, strain and sex on performance, mortality and uniformity at 42 days of age (mean±SD)

	feed intake (1-42d) (g/d)	body weight d 42 (g)	FC (1-42d)	total mortality (%)	SDS (%)	uniformity
feed restriction						
ad lib	90.9±5.1	2220±168	1.755±0.053	8.5±2.8	2.7±2.3	87.7±20.6
low energy[5]	90.0±5.4	2213±187	1.744±0.051	8.9±3.9	1.7±1.8	86.6±8.0
NaCl	90.8±5.0	2229±168	1.745±0.047	8.1±4.3	1.7±1.8	92.6±5.5
strain						
Ross	90.4±4.8	2228±152	1.740±0.041	9.1±3.9	2.8±2.2[a]	85.6±17.2
Hybro	90.6±5.4	2211±189	1.756±0.056	7.9±3.4	1.2±1.4[b]	92.3±5.5
sex						
male	95.3±1.6[a4]	2378±63[a]	1.713±0.032[a]	10.1±3.5[a]	2.7±2.2[a]	85.6±16.6
female	85.8±1.5[b]	2061±45[b]	1.783±0.035[b]	6.9±3.1[b]	1.4±1.5[b]	92.3±7.1
ANOVA (*p*-values)						
feed restriction (A)	0.272	0.752	0.672	0.843	0.239	0.434
Strain (B)	0.540	0.345	0.079	0.285	0.011	0.098
Sex (C)	0.000	0.000	0.000	0.011	0. 041	0.108
A x B	0.030					

[4] means with the same letter are not significantly different from each other ($p<0.05$)
[5] 'low energy': low energy, normal protein diet from d 4 for 4 d
'NaCl': diet with low NaCl content (0.05 %) from d 4 for 4 d

3.2. Compensatory growth

Using the Gompertz equation, daily growth is estimated in function of time for the different dietary treatments within each strain and for both sexes (Figure 22-23). Qualitative feed restrictions induced a pronounced compensatory growth on Ross broilers (Figure 22). In result, restricted Ross male birds were heavier than the control group (low energy: +51 g; NaCl: +73 g). These differences were statistically not significant.

The ‘catch-up’-growth for the Ross females started somewhat later (Figure 22). The ‘low energy’-treatment resulted in an equal final body weight as the control. On the other hand, the more pronounced ‘catch-up’ for the ‘NaCl’-treatment explains the higher final body weight for these birds in comparison with the control birds (20 g higher).

As the compensatory growth for the restricted Hybro males was only limited or even non-existing, broilers were not able to catch up completely with the control group (Figure 23). Controls reached a final body weight of 2420 g while ‘low energy’-chickens weighed 2384 g and ‘NaCl’-chickens 2357 g (not significantly different). Hybro females showed a more pronounced ‘catch-up’-growth. Again the differences were not significant. The ‘NaCl’-treatment resulted in a mean final body weight comparable with the non-restricted chickens (2051 g). The compensatory growth after the ‘low energy’-treatment started a few days later and was insufficient to catch up completely (2004 g).

3.3. Mortality and uniformity

Total mortality (dead + removed) was not significantly influenced by treatment (Table 17). Male broilers knew a significantly higher mortality in comparison with females. No significant strain effect was found.

Only a positive trend for reduction of the cases of SDS by restriction was found (Table 17). In general, it appears that Ross birds or male chickens were the most sensitive for SDS. During the entire trial, only 6 birds were removed with leg problems and 4 chickens had ascites as cause of death. Uniformity was not impaired by qualitative feed restrictions. Hybro chickens showed a trend for a higher uniformity in comparison with Ross birds.

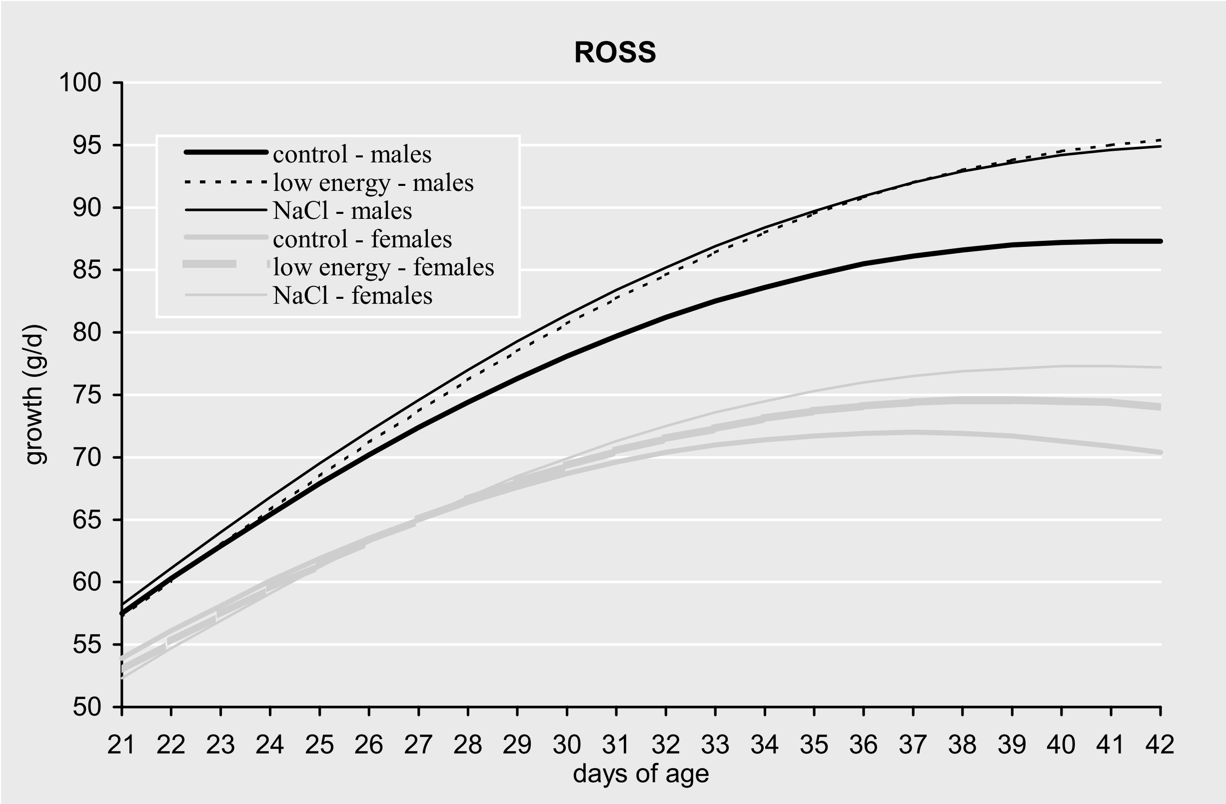

Figure 22 : Effect of feed restriction on growth Ross males and females between 21 and 42 days of age

Males: control : $W_t = 41.9\ e^{(0.1962/0.0395)(1-e^{-0.0395t})}$
low energy : $W_t = 41.9\ e^{(0.1906/0.0372)(1-e^{-0.0372t})}$
NaCl : $W_t = 41.9\ e^{(0.1931/0.0379)(1-e^{-0.0379t})}$

Females: control : $W_t = 41.7\ e^{(0.2004/0.0426)(1-e^{-0.0426t})}$
low energy : $W_t = 41.7\ e^{(0.1951/0.0408)(1-e^{-0.0408t})}$
NaCl : $W_t = 41.7\ e^{(0.1913/0.0394)(1-e^{-0.0394t})}$

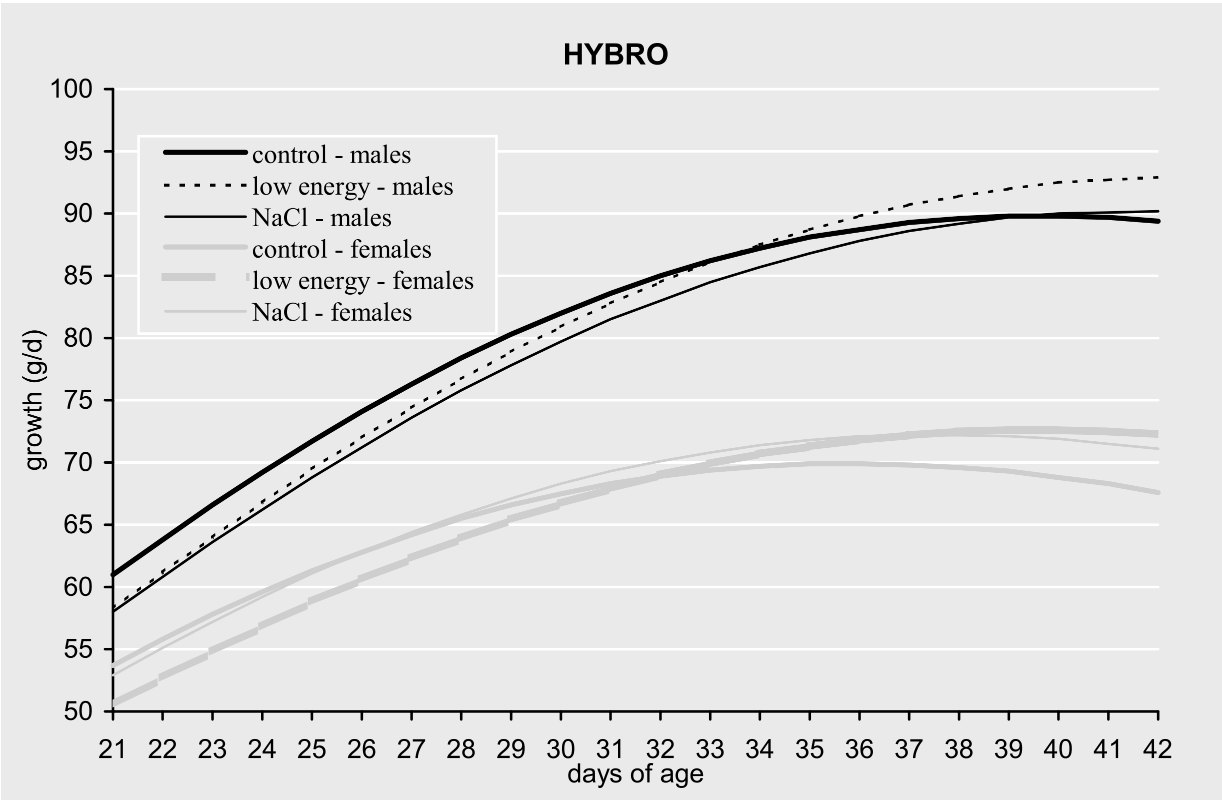

Figure 23 : Effect of feed restriction on growth Hybro males and females between 21 and 42 days of age

Males: control : $W_t = 40.1\ e^{(0.2053/0.0411)(1-e^{-0.0411t})}$
low energy : $W_t = 40.1\ e^{(0.1975/0.0388)(1-e^{-0.0388t})}$
NaCl : $W_t = 40.1\ e^{(0.1985/0.0393)(1-e^{-0.0393t})}$

Females: control : $W_t = 39.9\ e^{(0.2054/0.0438)(1-e^{-0.0438t})}$
low energy : $W_t = 39.9\ e^{(0.1935/0.0402)(1-e^{-0.0402t})}$
NaCl : $W_t = 39.9\ e^{(0.2006/0.0421)(1-e^{-0.0421t})}$

3.4. Carcase composition

Restrictions or strain did not change total protein, total lipid and abdominal fat content (Table 18). As to be expected, fat deposition was lower for male broilers than female chickens. However, it could not be illustrated that male broilers also had a higher protein deposition.
After slaughter, carcases of chickens previously restricted, showed the same meat yield as the controls (Table 18). Their breast meat proportion even tended to be higher. Ross chickens produced 1.3 % more breast meat than Hybro chickens. Female broilers had 0.5 % more breast meat in comparison with males.

Table 18 : Effect of qualitative feed restriction, strain and sex on protein and lipid content (g/kg whole bird inclusive feathers), abdominal fat content, carcase yield and breast meat percentage (42 days of age) (mean±SD)

	protein content	lipid content	abd. fat (g/kg)	yield (%)	breast meat (%)
feed restriction					
ad libitum	185.5±3.2	136.4±14.2	20.4±4.0	67.1±0.3	27.3±0.9
low energy[6]	187.6±5.0	138.2±14.3	19.0±3.3	67.4±0.7	27.9±0.9
NaCl	186.0±4.9	138.3±12.0	19.2±3.4	67.2±0.5	27.7±0.9
strain					
Ross	186.6±4.3	137.4±13.7	19.8±3.4	67.3±0.5	28.3±0.7[a]
Hybro	186.1±4.7	137.9±13.1	19.2±3.8	67.1±0.6	27.0±0.7[b]
sex					
male	186.9±3.7	127.7±9.1[a7]	16.6±1.4[a]	67.3±0.6	27.4±1.0[a]
female	185.8±5.1	147.6±8.3[b]	22.4± 2.4[b]	67.1±0.4	27.9±0.7[b]
ANOVA (*p*-values)					
feed restriction. (A)	0.518	0.872	0.255	0.343	0.118
strain (B)	0.769	0.869	0.374	0.277	0.000
sex (C)	0.486	0.000	0.000	0.307	0.048

3.5. Meat quality

Treatment, strain or sex had only a minor effect on meat quality parameters (Table 19). The ‘low-energy’-treatment showed a trend of and increased b-value. The opposite counted for the ‘NaCl’-treatment. The interaction for drip indicated the variable effect of the treatments according to strain and sex. The significant difference between sexes had mainly to do with a

[6] ‘low energy’: low energy, normal protein diet from d 4 for 4 d
‘NaCl’: diet with low NaCl content (0.05 %) from d 4 for 4 d
[7] means with the same letter are not significantly different from each other ($p<0.05$)

pronounced difference for the restricted Ross 508-birds. The a-value was significant higher for female broilers.

Table 19 : Effect of qualitative feed restriction, strain and sex on meat quality (mean±SD)

	pH_{24}	colour			moisture	drip	cook. losses	shear force
		L	a	b	(%)	(%)	(%)	(N)
feed restriction								
ad libitum	5.8±0.1	57.2±2.3	7.3±0.9	14.0±2.3[ab8]	27.7±2.6	6.4±1.8[a]	13.6±2.4	12.8±2.6
low energy[9]	5.8±0.1	56.3±2.1	8.0±1.5	15.0±1.3[b]	26.7±2.2	6.9±2.5[ab]	12.1±2.4	14.5±4.2
NaCl	5.9±0.1	56.7±2.9	7.4±1.7	12.9±1.2[a]	25.6±2.7	7.9±2.6[b]	11.8±1.9	12.4±3.9
strain								
Ross	5.8±0.1	56.3±2.5	7.5±1.4	14.0±1.8	26.1±1.9	6.7±1.9	11.8±2.2	13.7±4.2
Hybro	5.8±0.1	57.1±2.3	7.6±1.4	14.1±2.0	27.2±3.2	7.5±2.7	13.2±2.3	12.8±2.9
sex								
male	5.8±0.1	56.5±2.1	7.0±1.1[a]	13.5±2.1	25.7±2.1	5.8±1.6[a]	11.9±2.3	12.5±4.1
female	5.8±0.1	56.9±2.7	8.1±1.5[b]	14.5±1.6	27.6±2.8	8.4±2.3[b]	13.1±2.3	14.0±3.1
ANOVA (p-values)								
feed restriction	0.357	0.715	0.459	0.063	0.240	0.105	0.158	0.376
strain (B)	0.674	0.452	0.814	0.789	0.297	0.143	0.086	0.509
sex (C)	0.093	0.747	0.048	0.139	0.063	0.000	0.159	0.259
A x B						0.033		
A x B x C						0.022		

4. Discussion

Results show that the qualitative feed restrictions used were effective ways to induce a compensatory growth after the temporary growth retardation. Final body weights of the chickens subjected to these restrictions showed no significant differences with the control birds.

However, by limiting early growth by reducing the energy content of the diet, it is sometimes difficult to predict the results. With lowering the energy level of the diet, an increased feed intake should be expected as the bird tries to keep his caloric intake on level. Indeed, Griffiths *et al.* (1977) and Leeson *et al.* (1996) noticed these increases in feed intake although these were insufficient to keep up with the energy intake of the control birds. In the present trial energy-restricted chickens consumed significantly less feed than the control birds during the period of restriction (12 % on average). This means that real energy deprivation was much

[8] means with the same letter are not significantly different from each other (p<0.05)
[9] 'low energy': low energy, normal protein diet from d 4 for 4 d
'NaCl': diet with low NaCl content (0.05 %) from d 4 for 4 d

higher than the initial 20 %. On the other hand, until 8 d of age, the low energy feed was used more efficiently than the ‘NaCl’-diet.
The (unexpected) depression in feed intake with the ‘low energy’-diet in the present trial has probably to do with the high degree of satiation and a negative influence on palatability, because of the high concentrations of oat hulls. However, in the trials of Zubair and Leeson (1994a) and Leeson *et al.* (1996) even higher concentrations of oat hulls were used and still chickens were able to adjust their feed intake. This apparent contradiction may have to do with the fact that these authors used corn based, pelleted diets while wheat was the main ingredient of the meal diets in the present trial. Maybe, when diets would have been crumbled, feed intake would have been less impaired (Newcombe and Summers, 1985).
In contrast with quantitative feed restriction where FC improved during and immediately after restriction (Scheideler and Baughman, 1993; Saleh *et al.*, 1996; Lippens *et al.*, 2000), in the present trial FC of feed restricted birds deteriorated during the period of restrictions. However, at the end of the trial there was a minor trend towards an ameliorating FC for the restricted birds. Meluzzi *et al.* (1995) demonstrated a significantly better FC at the age of 49 d when growth was restricted with a low sodium concentration in the feed from 7 to 14 d (Na = 0.02%). This amelioration of the FC on d 42 was, in accordance with the present results, not significant. No results at the age of 42 d are available in the study of Plavnik and Hurwitz (1990) using a low sodium diet (Na = 0.03%) for 6 d. These authors were able to demonstrate a significantly better FC at the age of 56 d, with no difference in final body weight. It must be noticed however that in the present trial a combination of low Na and low Cl was used, while in the mentioned studies only Na was lowered. On the other hand, when the present trial was prolonged, the amelioration of the FC of restricted birds might also have proceeded to become significantly better.
It is seen many times in literature that a lot of factors influence the initiation and the magnitude of the established compensatory growth. Again this was confirmed in the present trial. Remarkable here was the pronounced compensatory growth of the Ross males. After quantitative feed restrictions from d 4 for 4 d with Ross 508 chickens, no compensatory growth was seen during the entire duration of the trial (6 weeks) in the previous results (Lippens *et al.*, 2000). After the qualitative restrictions in the present trial however, Ross 508 males were able to establish a pronounced and early ‘catch-up’ growth. It is doubtful that these quite different results are mainly attributable to the kind of restrictions (qualitative or quantitative) because both kind of restrictions induced a similar growth retardation and

conditions during realimentation were similar. Rather initial chicken quality than restriction method seems to be of major importance for the ability to establish compensatory growth. Indeed, in the trial of Lippens *et al.* (2000) initial body weight was only 38.1 g on average. One day old Ross 508 chickens weighed on average 41.8 g in the present trial. This might explain partly the difference in results (see also Lippens *et al.*, 2002a). More research is needed on this subject.

No clear effect of qualitative feed restrictions on mortality could be demonstrated. However, in accordance with the results from Lippens *et al.* (2000), all treatments had a rather positive effect on the fall-out because of SDS. Probably, the relative low number of broilers used in these kind of trials can explain the missing of significant differences on mortality.

Also of great economic interest is the fat deposition in broiler chickens. In contrast to the results of Plavnik and Hurwitz (1985, 1988, 1991) and Jones and Farrell (1992a), no reduction in abdominal fat content was found. In the trial of Lippens *et al.* (2000) also a quantitative feed restriction could not lower abdominal fat contents.

The dressing yield of the carcases was not affected by treatment. It may be concluded that when restrictions are used which do not reduce final body weight significantly, no loss in carcase yield should be expected (Leeson *et al.*, 1991; Scheideler and Baughman, 1993; Zubair and Leeson, 1994a; Lippens *et al.*, 2000). In contrast with these results, Saleh *et al.* (1996) found an improvement in dressing percentage while final body weight was significantly reduced.

For the influence of feed restrictions on yield of breast meat, it is difficult to see a clear picture. Most of the results in literature cannot demonstrate a significant effect of feed restrictions on breast meat proportion (Pinchasov and Jensen, 1989; Plavnik and Hurwitz, 1990; Leeson *et al.*, 1991; Van Harn, 1992; Carter *et al.*, 1994; Zubair and Leeson, 1994a; Palo *et al.*, 1995; Lippens *et al.*, 2000) which is in line with the present results. Inducing a significant improvement of the breast meat proportion with feed restrictions is rarely seen in literature (Saleh *et al.*, 1996). In contrast, when restrictions are rather severe, lower breast meat proportions can be expected (Lippens *et al.*, unpublished; McGovern *et al.*, 1999). In contrast with the results of Hopić *et al.* (1997) and Lippens *et al.* (2000), sex had a significant effect on breast meat yield. In the technical brochure of Ross 508-chickens (Ross Breeders, 2000) however differences between the female and the male breast percentages are not that pronounced.

The significant effect of genotype on cut-up parts was expected, as the Ross 508 strain is selected for high breast meat proportions. This agrees with the results of Rémignon *et al.* (1996) and Lippens *et al.* (2000). However, according to these researchers and the present trial results, the impact on meat quality of these genetic differences between strains is almost non-existing.

In contrast, according to Dransfield and Sosnicki (1999), higher growth rates may induce morphological abnormalities, induce larger fibre diameters and a higher proportion of glycolytic fibres together with a lower proteolytic potential in the muscles. This means that, after death, a faster development of the rigor mortis increases the likelihood of paler poultry meat, reduced water holding capacity and increased toughness of poultry meat. No significant prove is found in the present trial to confirm this theory. Moreover, it is questionable that the small differences found in the present trial are of major importance for the organoleptic perception of the meat. Indeed, the differences were very small and the correlation between organoleptic perception by the consumer and these instrumental measurements is found to be rather low (Van Oeckel, 1999). In other words, there is no indication in the present trial that changes in growth pattern (growth retardation followed by compensatory growth) induced by a qualitative feed restriction, were of that nature that meat quality was impaired.

In conclusion, qualitative feed restrictions as used in the present trial, gave some indications of being a practical tool to reduce losses due to metabolic diseases without deteriorating performance parameters. No indications of negative effects on carcase composition or meat quality were found.

The question arises, however, if these quantitative feed restrictions are really a practical alternative for the quantitative feed restriction programmes mentioned before. It can indeed mean an extra cost to provide a quantity of feed of a lower quality. Supplemental stocking place and distribution possibilities have to be available. Moreover, it concerns only very low amounts, which can mean an extra cost for delivery on the farm. When using qualitative feed restriction, using a diet with a lower concentration of one or more essential nutrients, the birds will increase their feed intake in an attempt to maintain nutrient intake. In this way, the method has the disadvantage that the effects may become rather unpredictable.

Broiler feed intake control (quantitative restriction), however, should not bring about many practical problems when considering the usually well-equipped broiler houses (automatic feed weighing and distribution). For these reasons, it was chosen to use quantitative feed restriction programmes only in the research described below.

Chapter 6

N-RETENTION DURING COMPENSATORY GROWTH

Adapted from :

Lippens, M., Huyghebaert, G. & De Groote, G. (2002). The efficiency of nitrogen retention during compensatory growth of food-restricted broilers. British Poultry Science 43 : 669-676.

ABSTRACT

1. Two experiments were conducted to investigate the influence of compensatory growth, induced by early quantitative feed restrictions, on the efficiency of nitrogen-retention in two lines of Ross broilers.
2. Birds were restricted to 80 % of the *ad libitum* intake of the previous 24 h of the control group from d 4 to d 7. After the period of restriction all birds were fed *ad libitum*.
3. In both trials, the final body weight of the restricted Ross 208(308) birds was similar or even higher than that of the control group. For the Ross 508 line, compensatory growth was substantial in the first trial, but non-existing in the second trial. In all cases, the mortality of restricted birds was lower than in control birds.
4. Not only environmental factors, but mainly chick quality, seemed to have a major influence on the capacity of the chickens to establish compensatory growth.
5. As compensatory growth was established, some improvement in N-retention was induced. Although differences were not significant, they can be important for the environment.
6. It is concluded that a retardation of the early growth of fast growing broiler chickens can, in certain circumstances, reduce mortality and increase performance and N-retention.

1. Introduction

Modern broiler chickens are characterised by high growth rates, which have been associated with increased fat deposition and high incidences of skeletal and metabolic diseases. Studies have shown that restricted growth early in life, followed by compensatory growth, can prevent these problems without reducing performance (Plavnik and Hurwitz, 1985, 1988, 1991; Robinson *et al.*, 1992; Saleh *et al.*, 1996; Lippens *et al.*, 2000). Better feed conversion have been observed (Plavnik and Hurwitz 1985, 1988, 1991).

Protein is one of the most expensive elements in the cost of a complete feed. It carries a second cost, as excreted nitrogen (N) contributes to the pollution of the environment. Thus it is important to use the feed protein as efficiently as possible. Feed restriction contributes to this by reducing protein intake in the early phases of growth when the concentration of protein is high. If there is compensatory growth, catch-up occurs during phases when demands relative to energy become low and higher efficiencies are expected. Indeed, Jones and Farrell (1992a); Buyse et al. (1994a, 1996) and Leeson and Zubair (1997) found indications of an amelioration of N-retention during the phase of compensatory growth. The restrictions conducted in the trial of Leeson and Zubair (1997), however, are economically not realistic because they are too severe to allow normal final body weights. Little is known about N-retention as a function of age, especially in feed-restricted birds.

Two trials were conducted to investigate how far N-retention in two lines of Ross broilers can be ameliorated during the compensatory growth induced by an economically feasible quantitative feed restriction.

2. Materials and methods

2.1. Experimental design

Both trials were designed as 2-factorial (two dietary treatments x two lines of broilers). In each trial, the first treatment consisted of a control group, with *ad libitum* feeding. Based on the results of Lippens *et al.* (2000) and some unpublished data, it was found that a restriction to 80 % of the *ad libitum* intake of the previous 24 h of the control group, from d 4 until d 7, is a good way to induce compensatory growth without loss in performance.

In both trials unsexed chickens were reared in pens of 100 chickens up to 42 d of age. Trial 1 consisted of 600 Ross 208 and 600 Ross 508 chickens, trial 2 of 600 Ross 308 and 600 Ross

508 chickens. Mean chick weights at day 0 in the first trial were 42.4 g (8.3 %) (mean (coefficient of variation of individual bird weights)) and 44.7 g (7.9 %) for Ross 208 and Ross 508-birds, respectively. In the second trial day-old weights were 40.1 g and 37.7 g (no individual bird weights available at day 0), respectively. In the first trial a high temperature schedule was used (starting at 36 °C and slowly decreasing to a constant temperature of 22 °C at 37 d of age). In the second trial, as in practice mostly lower temperature schedules are used, ambient temperature was set at 30 °C for the first 3 d slowly decreasing to 17 °C at d 42.

2.2. Diets

Except for the period of feed restriction, the birds were fed *ad libitum*. In both trials, a starter diet with 225 g CP/kg and 12.42 MJ AME_n (broilers; CVB 1997)/kg was given until 14 d of age. From d 15 until d 42 a grower diet with 200 g CP/kg and 12.85 MJ AME_n/kg was offered. For the ingredient composition and the calculated chemical analysis see Table 20.

2.3. Response parameters

General response parameters are mentioned in Chapter 3. Every week (d 0, d 8, d 14, d 21, d 28, d 35 and d 42) 6 chickens per pen (3 males, 3 females), were used for lipid and protein contents (pooled samples per pen for each replicate). Nitrogen-retention was calculated as the ratio of the amount of N retained in the body to N-intake during the same period (‘comparative slaughter technique’).

3. Results

3.1. Trial 1

Compensatory growth occurs when chickens whose growth has been retarded by dietary restriction grow at a faster rate than animals of the same age which have not had any prior restriction. Feed restriction to 80 % during 4 d starting at d 4 induced an average growth retardation of 8.3 % at the age of 8 d (results not shown). However, compensatory growth was induced, which made it possible to exceed the final body weight of the *ad libitum* fed broilers (Table 21). As feed intake only marginally increased, restricted birds converted their feed more efficiently in comparison with the controls. As well as greater efficiency, there was a positive effect of feed restrictions on mortality (p= 0.073).

Lipid content of the whole bird was, however, not changed by dietary treatment. Ross 508 birds reached a somewhat higher final body weight than the Ross 208 birds. Similar feed conversions were recorded. Total lipid content (inclusive of feathers) was higher in the 508 birds. This difference however was not significant.

Table 20 : Ingredient composition and calculated nutrient composition of the diets (g/kg, unless otherwise stated)

	Trial 1		Trial 2	
Ingredients	**starter[1]**	**grower[2]**	**starter[3]**	**grower[4]**
Wheat	400.0	500.0	400.0	500.0
Soybeans (full fat)	60.5	129.6	11.2	61.1
Soybean meal (44 % CP)			167.8	111.2
Soybean meal (48 % CP)	249.9	134.6	105.8	73.9
Yellow corn	137.7	100.0	150.1	100.0
Fish meal	17.0	23.2	50.0	30.0
Animal fat	80.0	68.7	70.0	67.8
Soybean oil			10.0	12.2
Tapioca		2.2		3.4
Dicalc. phosph. .2H_2O	16.6	12.0	12.9	11.3
Limestone	7.7	10.4	7.2	10.2
Sodium chloride		2.21	1.9	2.1
Sodium bicarbonate	5.5	2.0	1.5	1.9
Vitamin/trace min. mix	10.0	10.0	10.0	10.0
DL-methionine	5.4	1.9	1.4	1.9
L-lysine-HCl	8.9	2.4		2.3
L-threonine	0.4	0.4		0.4
Biofeed + CT	0.4	0.5	0.4	0.5
Nutrients (g/kg) (calculated)				
CP	225.0	199.4	225.0	199.8
AME_n, MJ/kg	12.42	12.85	12.42	12.85
$Isoleucine_{ad}$	8.4	7.5	8.8	7.5
$Leucine_{ad}$	14.2	12.7	15.0	12.8
$Lysine_{ad}$	17.0	10.7	10.9	10.7
$Meth._{ad}$ + $Cyst._{ad}$	11.3	7.5	7.9	7.5
$Phenyl._{ad}$ + $Tyr._{ad}$	15.1	13.5	15.6	13.5
$Threonine_{ad}$	7.2	6.5	7.3	6.5
$Tryptophan_{ad}$	2.2	2.0	2.3	2.0
$Valine_{ad}$	9.0	8.1	9.5	8.2
$Arginine_{ad}$	12.1	10.7	12.5	10.7
$Histidine_{ad}$	4.5	4.1	4.8	4.1
$NEAA_{ad}$	94.8	98.5	94.8	87.2

Although there was no significant interaction between treatment and line it is still interesting to look at the performance of the separate lines. Both lines showed similar growth retardation

[1] containing 100 mg/kg monensin and 0.04 g/kg STAFAC (virginiamycine-50 %)
[2] containing 1 mg/kg diclazuril and 0.04 g/kg STAFAC (virginiamycine-50 %) (except for the last 5 d)
[3] containing 100 mg/kg monensin and 5 mg/kg flavomycine
[4] containing 1 mg/kg diclazuril and 5 mg/kg flavomycine (except for the last 5 d)

due to restriction (results not shown). Results shown in Table 22, however, show that compensatory growth was most pronounced with the Ross 508 broilers, resulting in almost significant improvements in the final body weight of the restricted birds. On average, these birds were 78 g heavier with an amelioration in FC of 0.042. Only in the 208 line, feed restriction slightly reduced lipid content (p=0.423). However, a trend towards elevation of lipid content was observed in the restricted broilers of the 508 line.

Table 21 : Effect of feed restriction and line on performance at 42 d of age (mean+SD, N=6) (Trial 1)

	feed intake (g/d)	**body weight (g)**	**FC**	**uniformity (%)**	**mortality (%)**	**lipid content**
treatment						
ad libitum	86.6±2.4	2117±63	1.754±0.015[a5]	46.8±4.0	5.5±2.2	133.9±10.2
restricted	86.9±2.2	2154±55	1.730±0.019[b]	49.2±5.0	3.2±1.3	134.7±12.3
line						
Ross 208	86.2±2.3	2120±59	1.743±0.015	47.5±4.2	4.5±1.6	131.7±11.7
Ross 508	87.3±2.1	2151±62	1.742±0.027	48.5±5.0	4.2±2.6	136.9±10.1
ANOVA (*p*-values)						
treatment	0.814	0.303	0.027	0.435	0.073	0.903
line	0.441	0.392	0.940	0.734	0.776	0.432

Table 23 shows the N-retention and body growth (weekly and total), for each line, as influenced by treatment. As there were significant interactions between treatment and line, a separation of the data between lines is justified. In both lines, restriction tended to enhance the overall N-retention. Although weight gain of restricted 508-birds was near to significantly higher than the controls, the improvement of the N-retention had a *p*-value of only 0.301. Besides, it was substantially less than the difference in N-retention within the 208 line.

For both lines during the first week (with feed restriction), the available protein was converted more efficiently into body protein by the restricted birds. For the 208 line, compensatory growth was noticed at weeks 4 and 6. Also in these two weeks a more efficient protein deposition was established (differences not significant). Birds of the 508 line showed a compensatory growth during the entire growth period (with the exception of week 5). There was higher N-retention during weeks 2, 3 and 5. During week 6 there was a similar N-retention in the two treatments despite the higher growth rate of the retarded chickens. Only week 4 showed a lower N-retention in the feed-restricted birds even though growth rate was slightly higher.

[5] means with a different letter are significantly different from each other ($p<0.05$) (within each factor)

Table 22 : Influence of feed restriction on performance of two lines of Ross broilers at the age of 42 d (mean+SD, N=3) (Trial 1)

	feed intake (g/d)		body weight (g)		FC		uniformity (%)		mortality (%)		lipid content	
	Ross 208	**Ross 508**	**Ross 208**	**Ross 508**	**Ross 208**	**Ross 508**	**Ross 208**	**Ross 508**	**Ross 208**	**Ross 508**	**Ross 208**	**Ross 508**
ad libitum	86.4±2.8	86.8±2.5	2122±75	2112±66	1.746±0.018	1.763±0.005[a6]	45.7±4.7	48.0±3.6	5.3±1.5	5.7±3.1	136.1±11.5	131.8±10.7
restriction	86.0±2.3	87.9±2.0	2119±56	2190±26	1.739±0.013	1.721±0.021[b]	49.3±3.5	49.0±7.0	3.7±1.5	2.7±1.2	127.4±12.4	142.0±8.0
***p*-value**	0.853	0.586	0.954	0.131	0.654	0.028	0.341	0.837	0.252	0.187	0.423	0.253

[6] means with a different letter are significantly different from each other ($p<0.05$) (within each factor and line)

Table 23 : Effect of feed restriction and line on the weekly growth (g) and N-retention (%) (mean+SD, N=3) (Trial 1)

	week 1	week 2	week 3	week 4	week 5	week 6	total
ROSS 208							
ad libitum							
growth	103.3±9.3	175.0±3.6	263.0±4.6	431.3±18.1	530.0±19.9	576.7±43.1	2079±75
N-retention	52.8±1.2	43.6±1.4	53.5±10.4	52.2±1.7	51.0±2.4	43.6±5.2	48.6±1.0
restricted							
growth	89.0±9.3	173.3±2.1	261.7±13.6	454.7±26.7	495.3±10.0	602.7±25.4	2077±56
N-retention	55.8±2.3	41.3±2.9	53.7±2.2	56.0±2.1	48.9±4.4	49.9±7.4	50.7±1.6
ROSS 508							
ad libitum							
growth	99.3±13.1	171.7±10.8	257.7±5.9	428.3±21.7	528.3±8.1	582.3±29.5	2067±67
N-retention	49.3±2.2	42.1±1.4[a7]	46.0±5.5	59.9±9.6	47.9±5.8	44.8±3.3	48.5±0.6
restricted							
growth	90.7±6.0	191.3±10.1	273.3±13.9	439.0±9.6	528.0±18.2	623.0±8.9	2145±25
N-retention	53.0±3.2	45.8±1.0[b]	54.5±1.8	55.0±2.8	50.0±4.0	45.0±5.4	49.6±1.4

3.2. Trial 2

In contrast with the observations in trial 1, compensatory growth was not found in trial 2 (Table 24). On average, restricted birds were 47 g lighter than their control counterparts. Food conversion and total lipid content did not change. However, some significant interactions were observed. These interactions become clear in Table 25.

Table 24 : Effect of feed restriction and line on performance at 42 d of age (mean+SD, N=6) (Trial 2)

	feed intake (g/d)	body weight (g)	FC	uniformity (%)	mortality (%)	lipid content
treatment						
ad libitum	93.4±2.3[a8]	2290±55	1.743±0.028	53.7±7.1	5.5±3.4	130.0±5.4
Restricted	91.6±3.3[b]	2243±115	1.747±0.033	51.2±6.0	2.9±2.0	133.3±1.0
line						
Ross 308	94.9±1.1[a]	2334±29[a]	1.738±0.027	50.5±5.8	3.6±2.7	132.1±3.6
Ross 508	90.1±1.7[b]	2199±76[b]	1.752±0.033	54.3±6.9	4.9±3.3	131.2±4.9
ANOVA (*p*-values)						
treatment	0.020	0.080	0.778	0.457	0.165	0.218
line	0.000	0.000	0.352	0.265	0.466	0.743
interaction		0.023	0.043			

Restricted Ross 308 birds had a higher final absolute body weight compared with the control groups (difference not significant). Some compensatory growth was established together with an amelioration of FC. Mortality was lowered by restriction. These results are comparable with the results of trial 1. The restricted 508 birds, however, had less ability to reach the target weight. They were 144 g lighter in comparison with the controls. Feed conversion

[7] means with a different letter are significantly different from each other (p<0.05) (within each factor and line)
[8] means with a different letter are significantly different from each other (p<0.05) (within each factor)

deteriorated by 4 %. Reduction in mortality after feed restriction was less pronounced than in the previous trial.

Significant interactions between treatment and line were found for weekly growth rate and N-retention. Separate results for the two lines are given in Table 26. In the 308 line, no overall improvement of the N-retention was observed. When looking at the results for each week, there were some phases where higher N-retention was seen in restricted birds. In contrast with the observations of trial 1, not during week 1 but during week 2 a better N-retention was obtained with the retarded birds. Compensatory growth seemed to start during week 4. Increase in retention was seen only during weeks 4 and 5.

Ross 508 birds showed no compensatory growth. There was greater N-retention only during the week of feed restriction. In consequence, overall N-retention was lower for the restricted birds.

4. Discussion

The retardation of initial growth rate, induced in the present trials by quantitative feed restriction, in some cases improved the performance of broiler chickens at the age of 42 d. This is in agreement with the results of Plavnik and Hurwitz (1985, 1988, 1991). However, these results were not a general rule. Within the 508 group of the second experiment, as in the study of Lippens *et al.* (2000), similar restrictions seemed to be too severe to allow a catch-up growth in comparison with the control birds. As there was no difference in response between the two trials for the 208/308-line, the different results may or may not be attributable to differences in environmental factors. The main reason for sometimes disappointing results is probably a combination of less favourable environmental conditions, health status of the birds and lower chick quality. Indeed, the 1-d-old 508 chickens in the second experiment had a mean body weight of only 37.7 g. This was lower than the 508 chickens of the first trial. Additional research is needed to investigate the influence of initial chick quality on the capacity of chickens to establish compensatory growth (see further).

Also the effect of dietary feed restriction on total carcase lipid content remains unclear. Differences in kind of broiler, environmental circumstances or perhaps rate of compensatory growth might be some of the reasons for variable results.

Table 25 : Influence of feed restriction on performance of two lines of Ross broilers at the age of 42 d (mean+SD, N=3) (Trial2)

	food intake (g/d)		body weight (g)		FC		uniformity (%)		mortality (%)		lipid content	
	Ross 308	**Ross 508**	**Ross 308**	**Ross 508**	**Ross 308**	**Ross 508**	**Ross 308**	**Ross 508**	**Ross 308**	**Ross 508**	**Ross 308**	**Ross 508**
ad libitum	95.3±1.0	91.5±1.0[a][9]	2325±13	2256±62[a]	1.753±0.027	1.732±0.031	48.3±0.6	59.0±6.2	5.4±2.8	5.6±4.5	131.1±5.2	129.0±6.6
restriction	94.5±1.2	88.8±1.1[b]	2344±42	2142±31[b]	1.722±0.017	1.772±0.024	52.7±8.3	49.7±3.8	1.8±0.9	4.1±2.2	133.1±1.3	133.5±0.8
***p*-value**	0.379	0.033	0.491	0.047	0.170	0.156	0.419	0.091	0.100	0.624	0.549	0.300

[9] means with a different letter are significantly different from each other ($p<0.05$) (within each factor and line)

Table 26 : Influence of feed restriction and line on the weekly growth (g) and N-retention (%) (mean+SD, N=3) (Trial 2)

	week 1	week 2	week 3	week 4	week 5	week 6	total
ROSS 308							
ad libitum							
growth	125.9±4.0[a][10]	209.7±5.9	335.7±15.9	463.7±16.6	557.3±9.7	592.0±16.4[a]	2284±12
N-retention	54.3±2.1	54.8±3.7	49.6±2.2	50.3±1.7	54.9±5.9	46.8±3.1	50.9±0.4
restricted							
growth	99.4±5.6[b]	209.0±11.	335.0±13.7	469.3±18.2	568.7±9.2	622.3±7.0[b]	2304±42
N-retention	52.7±2.2	56.3±5.0	47.1±5.0	55.4±2.8	55.7±2.5	44.5±2.4	51.0±1.2
ROSS 508							
ad libitum							
growth	116.7±1.6[a]	196.0±3.5	322.0±17.1	443.3±11.5	549.3±6.7[a]	590.7±30.9	2218±62[a]
N-retention	53.7±0.5	52.1±2.9	49.3±3.2	53.9±1.2	56.8±3.9	44.8±2.0	51.1±1.0[a]
restricted							
growth	92.4±8.2[b]	194.3±2.5	295.3±2.3	420.0±12.8	526.0±9.5[b]	576±21.7	2104±31[b]
N-retention	55.2±1.3	50.6±3.1	48.9±4.0	49.3±5.9	56.6±3.5	41.0±7.2	48.9±0.9[b]

Adequate compensatory growth seems to be correlated with an improvement of the N-retention of retarded birds in comparison with the *ad libitum* fed birds. Our results are in agreement with the findings of Leeson and Zubair (1997), where feed-restricted birds showed significantly higher N-retention during compensatory growth. Also chickens kept in an intermittent lighting schedule retained dietary protein during the catch-up phase more efficiently in comparison with chickens held in continuous light (Buyse *et al.*, 1994b, 1996). According to Buyse *et al.* (1996) and Kühn *et al.* (1996), the enhanced N-efficiency can be attributed to higher concentrations of circulating growth hormone (GH) and insulin-like growth factor-I during compensatory growth. Indeed, when there was no compensatory growth in restricted Ross males in the trial of Lippens *et al.* (2000), no significant differences in GH pulse parameters were detected (Govaerts *et al.*, 2000).

The improvement in N-retention in the present study was rather low in comparison with the results of Leeson and Zubair (1997). In this latter trial, however, feed restriction was very severe (50 % of the *ad libitum* intake for 6 d) giving a very strong growth retardation. The consequence was that a much higher compensatory growth was induced, combined with a higher N-retention (about 10 % from 13-19 d of age). However, such restrictions are economically undesirable because the final body weight of restricted birds is far below that of the control birds. Nevertheless, the current increases in N-retention can be of economical importance.

Also in the present trial it was found that kind of broiler chicken had its influence on the correlation between compensatory growth and N-retention. The 508 chickens needed more

[10] means with a different letter are significantly different from each other (p<0.05) (within each factor and line)

compensatory growth in comparison with the 208(308) line to realise the same degree of improvement in N-retention. The 508-chickens are genetically clearly different from the classic line and the observed differences might be related to a combination of the retarded juvenile growth rate and the higher breast meat percentage at slaughter age typical for this line.

There also seems to be an age-related variation in N-retention, regardless of treatment. Nitrogen retention is initially rather high and then drops to have a second maximum in weeks 4 and 5. Modern broiler lines are characterised by a very high early post natal growth rate (Ricklefs, 1985), which may be correlated with this high N-retention in the first week. The pronounced drop in trial 1 in comparison with the second trial during week 2 might have something to do with the rather high ambient temperature during this first half of the growing cycle, which is a possible stress factor for the birds. Also the relative high lysine concentration in the starter of the first trial may have induced a decrease in N efficiency. As the protein content of the grower is reduced after 14 d, efficiency can increase again. The decreasing N-retention at the end of the trial may result from the fact that with age, excesses of amino acids become higher resulting in a lower efficiency. More research is needed on this subject.

In conclusion, the possibility of a quantitative feed restriction to 80 % (from d 4 for 4 d) to induce compensatory growth depends on both the environmental circumstances and chicken quality. If compensatory growth can be established, hormonal changes may attribute to the amelioration of the N-retention in the chicken. Changing the growth curve of fast growing broiler chickens may be a way not only to improve performance but also to alleviate environmental pollution.

Chapter 7

INFLUENCE OF DIETARY PROTEIN CONTENT ON COMPENSATORY GROWTH CAPACITY

Adapted from :

Lippens, M. (2001). Influence of feed protein content on compensatory growth capacity and carcass composition of feed restricted broiler chickens. Poster presented on : 13th European Symposium on Poultry Nutrition, Blankenberghe, September 30 – October 4 : 39-40.

ABSTRACT

Quantitative feed restrictions are used to induce an early growth retardation (followed by compensatory growth) which makes it possible to control the high incidences of leg problems, metabolic diseases and increased fat deposition, common for our modern meat-type chickens. The objective of the present trial was to look at the influence of the grower protein content on the compensatory growth capacity and carcass composition of feed restricted broilers of two different Ross lines (Ross 308 and Ross 508). Furthermore, the influence of lowering the CP-content of the starter was examined.

The influence of feed protein content (starter or grower) is quite different between the two lines. Lowering the protein content of the starter diet of early-restricted birds can give good results on performance and carcass composition for both lines. However, especially Ross 508 chickens seemed to have their benefit of receiving a high protein grower after early feed restriction. The dietary grower protein content had positive effects on total carcase protein content and carcase yield. For both lines, a reduced mortality after feed restriction was difficult to indicate probably because of the rather limited number of birds in trial.

1. Introduction

Because modern meat-type chickens show a higher incidence of leg problems, metabolic diseases and increased fat deposition, a lower initial growth rate is pursued to avoid these negative selection responses. Early quantitative feed restriction seems to be a possibility to control these unwanted losses without significant reduction in final body weights (Plavnik and Hurwitz, 1991; Lippens *et al.*, 2000). However, little is known about protein (AA) requirement before and during the period of compensatory growth. Model-based calculations of Plavnik and Hurwitz (1989) show an increased requirement for most of the essential AA. Other authors could not show any positive effect of increased AA contents during 'catch-up' growth (Jones and Farrell, 1992a; Leeson and Zubair, 1997). The objective of the present trial was to look at the influence of the grower protein content on the compensatory growth capacity and carcass composition of feed restricted broilers of two different Ross lines.

Also, the influence of lowering the CP-content of the starter was examined. Results from Holsheimer *et al.* (1993) and Balbaie *et al.* (1999) showed that using low protein starter diets can give similar results in comparison with a normal starter diet. However, in the results of Colnago *et al.* (1991) a reduced feed intake and growth rate on the one hand and an increased FC and abdominal fat content on the other hand was found when the protein content of the starter was lowered from 23 to 18 %. These findings were confirmed by the work of Moran *et al.* (1991). It is however interesting to investigate if there exists an interaction between the protein content of the starter diet and the use of a quantitative feed restriction.

2. Materials and methods

2.1. Experimental design

A 3-factorial experiment (two starters x 3 grower treatments x two lines of broilers) was set up with 1630 Ross 308 (mean day-old weight : 43.8 with coefficient of variation of the total individual birds of 8.5 %) and 1630 Ross 508 (mean day-old weight : 42.6 with coefficient of variation of the total individual birds of 8.8 %) unsexed broiler chickens in order to investigate the effect of feed protein content on the capacity for catch up growth.

The two *ad libitum* treatments consisted of 3 replicates of 100 birds per pen. The other treatments had 2 replicates of 100 birds per pen and 2 replicates of 33 birds per pen. Final sex ratios (males/females) were 47/53 and 50/50 for the Ross 308 and Ross 508-chickens, respectively. Correction for differences in sex ratios were carried out as mentioned in chapter 3. A lighting schedule with increasing photoperiod (d 4-13: 6 h light, d 14-20: 10 h light, d 21-27: 14 h light, d 28-34: 18 h light; from d 35: 23 h light) was used. Average environmental air temperature started at 30 °C during the first 3 days to gradually decrease to 17 °C at the end of the trial.

2.2. Diets

Starter diets, with an energy content of 12.42 MJ AME_n, were given from d 0 until d 10. Grower diets, with an energy content of 12.85 MJ AME_n, were given from d 11 until 42 d of age. The following treatments were applied:

(1) normal protein starter (22 % CP)
- *ad libitum*-normal protein grower (20 % CP) **(*ad lib* NP)**
- 80 % of *ad libitum*-normal protein grower (20 % CP) **(80 % NP-NP)**
- 80 % *of ad libitum*-**high** protein grower (22 % CP) **(80 % NP-HP)**

2) low protein starter (20 % CP)
- *ad libitum*-normal protein grower (20 % CP) **(*ad lib* LP)**
- 80 % of *ad libitum* -normal protein grower (20 % CP) **(80 % LP-NP)**
- 80 % of *ad libitum*-**high** protein grower (22 % CP) **(80 % LP-HP)**

The crude protein content of all diets were formulated with respect for the ideal amino acid (AA) balance (Lippens *et al.*, 1997; Mack *et al.*, 1999). For the ingredient composition and the chemical analysis see Table 27.

2.3. Response parameters

General response parameters are presented in Chapter 3. At 43 d, 16 chickens per treatment were used to determine lipid and protein content.

3. Results

3.1. Performance and mortality

Tables 28 and 29 show the performance data for the entire period for each line. As the effects of line were significantly correlated with treatments, results are separated by line. Although body weight was not significantly changed with the dietary treatments (Tables 28-29 and Figures 24-25), still it seems that Ross 508 chickens had a higher capacity to compensate for growth retardation in comparison with 308 birds. Within the normal protein starter groups it probably had to do with the lower relative growth retardation of the 508 line at the age of 8 days (12.5 % against 8.5 % for Ross 308 and Ross 508, respectively), rather than differences in growth rate during the period of compensatory growth. However, within the low protein starter groups, the opposite counted for the relative growth retardation (10 % against 14 %) which made the catch up growth of the 508 line even more pronounced. Giving a high protein grower can further stimulate compensatory growth (Figures 26 and 27).

Table 27 : Ingredient composition and calculated nutrient composition of the diets (g/kg , unless otherwise stated)

Ingredients	**starter LP[1]**	**starter NP[1]**	**grower NP[2]**	**grower HP[2]**
Wheat	400.0	400.0	500.0	500.0
Soybeans (full fat)	61.3	66.5	136.0	55.8
Soybean meal (44 % CP)	22.4			
Soybean meal (48 % CP)	203.4	234.4	121.8	204.4
Fish meal		30.0	30.0	50.0
Yellow corn	190.9	159.7	100.0	71.5
Peas	3.8			
Tapioca			9.15	
Soybean oil				14.3
Animal fat	70.0	70.0	62.6	65.7
Dicalc. phosph. $2H_2O$	18.73	14.99	11.15	8.66
Limestone	7.93	7.63	10.33	10.15
Sodium chloride	2.42	2.25	2.05	1.46
Sodium bicarbonate	2.59	1.70	1.96	2.07
Vitamin/trace mineral mix	10.00	10.0	10.0	10.0
DL-methionine	2.46	1.69	1.88	2.26
L-lysine-HCl	2.68	0.55	2.28	2.35
L-threonine	1.05	0.17	0.38	0.55
Biofeed + CT	0.40	0.40	0.50	0.50
L-Arginine				0.29
Nutrients (g/kg) (calculated)				
CP	200.2	220.0	200.1	222.4
AME_n, MJ/kg	12.42	12.42	12.85	12.85
$Isoleucine_{ad}$	7.6	8.6	7.5	8.5
$Leucine_{ad}$	13.2	14.6	12.7	14.1
$Lysine_{ad}$	10.8	10.8	10.7	12.3
$Meth._{ad}$ + $Cyst._{ad}$	7.9	7.9	7.5	8.6
$Phenyl._{ad}$ + $Tyr._{ad}$	13.9	15.4	13.5	15.0
$Threonine_{ad}$	7.2	7.2	6.5	7.5
$Tryptophan_{ad}$	2.0	2.3	2.0	2.2
$Valine_{ad}$	8.1	9.2	8.2	9.3
$Arginine_{ad}$	10.9	12.3	10.7	12.3
$Histidine_{ad}$	4.1	4.7	4.1	4.6
$NEAA_{ad}$	87.5	94.8	86.8	96.4

[1] containing 100 mg/kg monensin and 5 mg/kg flavomycine
[2] containing 1 mg/kg diclazuril and 5 mg/kg flavomycine (except for the last 5 d)

Especially Ross 508 chickens fed a low protein starter seemed to have a higher need for protein during 'catch-up' growth. The slight trend for a worse FC after a lower dietary protein content during the starter period could be prevented by a quantitative feed restriction (Table 28-29 and Figure 25). A higher protein content of the grower could further ameliorate FC significantly.

The effect of dietary treatments on mortality is summarised in the Tables 28 and 29. The expected reduced mortality after early feed restriction was limited to a positive trend in the 308 line.

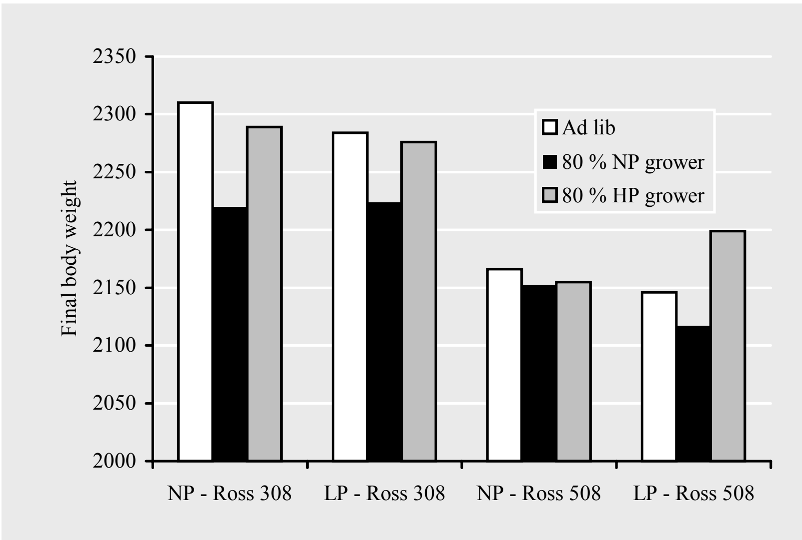

Figure 24 : Influence of feed restriction and feed protein content on final body weight of Ross 308 and Ross 508 broilers (42 d of age)

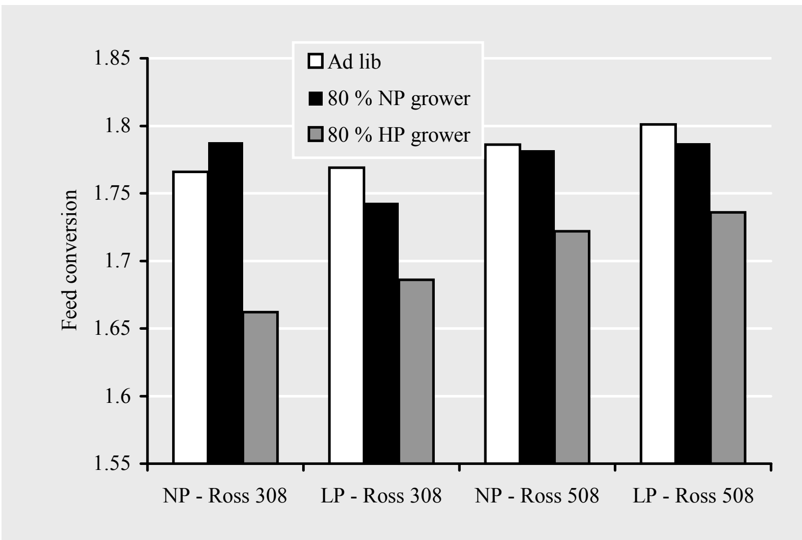

Figure 25 : Influence of feed restriction and feed protein content on FC of Ross 308 and Ross 508 broilers (42 d of age)

3.2. Carcase composition

The effect of dietary treatment on carcase composition was quite different between the broiler lines (Tables 30 and 31). For the Ross 308 birds, early feed restriction had a clearly higher impact on carcase quality than grower protein content. The early feed restriction had a significantly positive effect on total lipid content (g/kg whole bird) but only a limited positive effect on abdominal fat content and breast meat percentage.

Table 28 : Influence of feed restriction and feed protein content on performance of Ross 308 broilers (42 days of age) (mean±SD)

	feed intake (g/d)	body weight (g)	body weight gain (g/d)	FC	mortality (%)
ad lib NP	95.0±1.3[a3]	2310±58	53.9±1.3	1.766±0.043[a]	6.9±2.8[a]
80 % NP-NP	92.9±1.8[a]	2220±72	51.8±2.3	1.788±0.052[a]	4.3±3.4[ab]
80 % NP-HP	88.5±2.0[b]	2289±22	53.5±0.2	1.662±0.039[c]	4.3±2.2[ab]
ad lib LP	93.7±1.6[a]	2284±4	53.3±0.5	1.769±0.015[a]	5.4±3.3[ab]
80 % LP-NP	89.5±2.2[b]	2224±56	51.9±1.7	1.743±0.038[ab]	2.5±0.4[b]
80 % LP-HP	87.9±1.4[b]	2276±86	53.1±2.1	1.686±0.053[bc]	4.6±2.7[ab]
***p*-values**	0.000	0.235	0.225	0.004	0.417

Table 29 : Influence of feed restriction and feed protein content on performance of Ross 508 broilers (42 days of age) (mean±SD)

	feed intake (g/d)	body weight (g)	body weight gain (g/d)	FC	mortality (%)
ad lib NP	90.1±2.7[ab3]	2166±67	50.6±2.0	1.786±0.018[a]	5.1±2.9
80 % NP-NP	89.5±1.1[ab]	2152±29	50.2±1.0	1.782±0.025[a]	5.8±4.8
80 % NP-HP	86.6±2.3[a]	2155±20	50.3±1.7	1.722±0.016[b]	6.8±1.9
ad lib LP	90.5±3.6[b]	2146±120	50.1±2.9	1.801±0.035[a]	5.2±3.0
80 % LP-NP	87.4±1.2[ab]	2117±28	49.4±0.7	1.787±0.037[a]	5.8±4.0
80 % LP-HP	89.7±1.2[ab]	2199±46	51.3±1.0	1.736±0.013[b]	9.7±2.3
***p*-values**	0.128	0.607	0.598	0.003	0.438

[3] means with the same letter are not significantly different from each other (p<0.05)

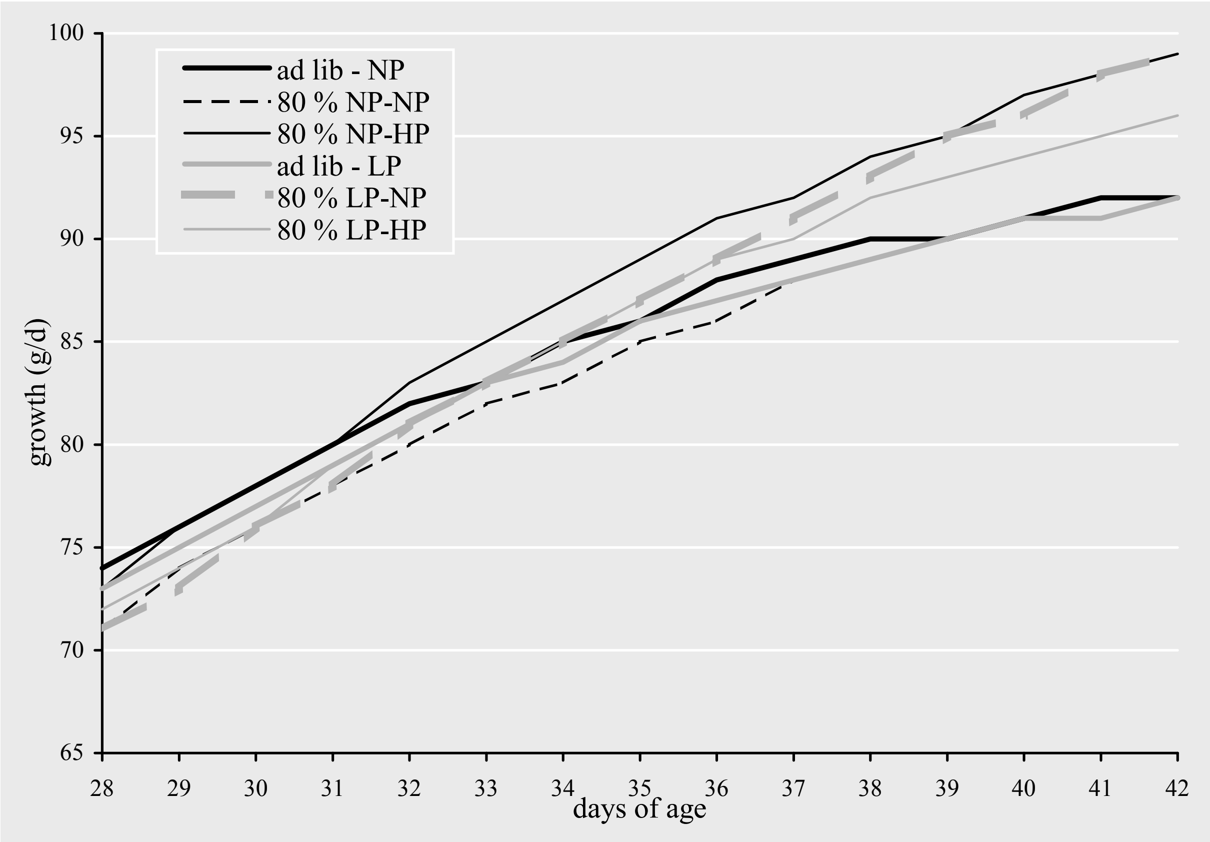

Figure 26 : Influence of feed restriction and feed protein content on growth performance of Ross 308 broilers (21-42d of age)

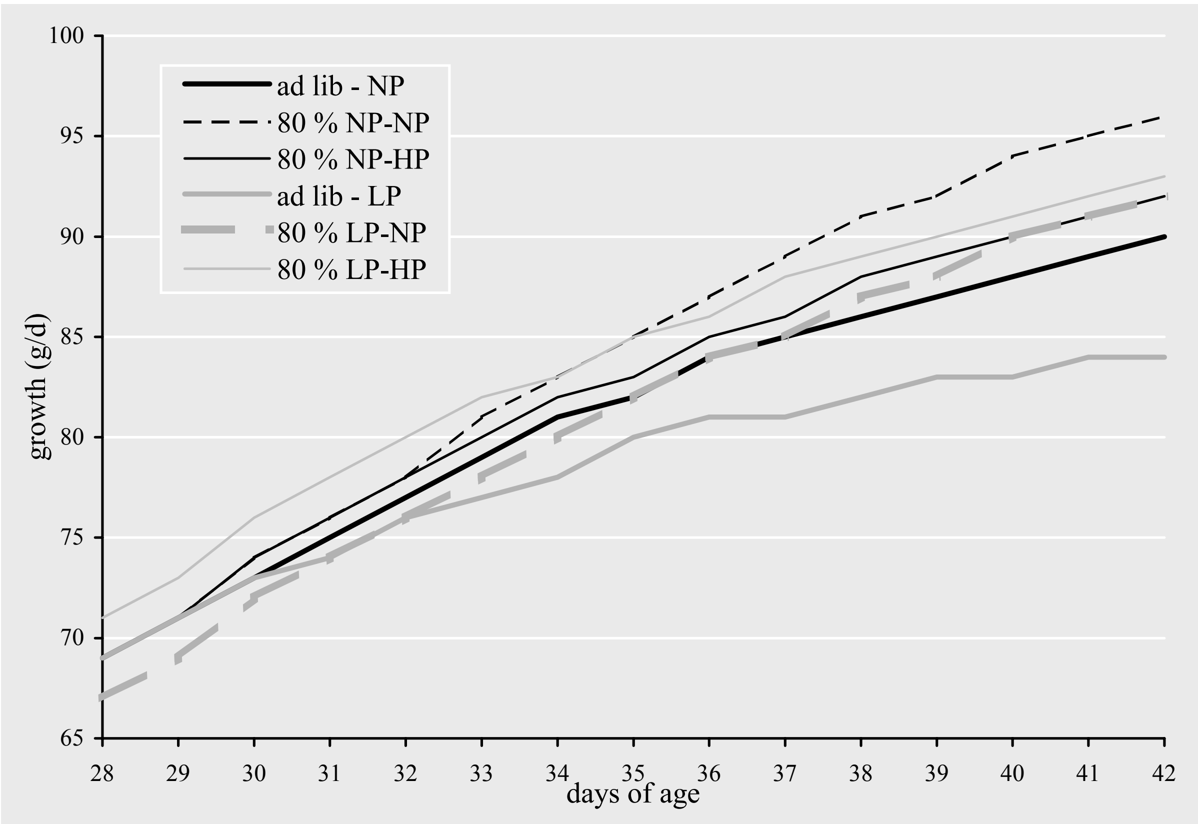

Figure 27 : Influence of feed restriction and feed protein content on growth performance of Ross 508 broilers (21-42d of age)

For the Ross 508 line, however, grower protein content had a clearly higher impact than feed restriction. The dietary grower protein content had positive effects on total carcase protein content ($p<0.05$) and carcase yield.

Table 30: Influence of feed restriction and protein content on protein and lipid content (g/kg whole bird inclusive feathers), abdominal fat content, carcase yield and breast meat percentage of Ross 308 broilers (42 days of age) (mean±SD)

	protein content	lipid content	abd. fat	yield (%)	breast meat (%)
ad lib NP	177.8±3.8	142.0±9.4[a4]	23.6±6.5	65.2±1.6	26.5±1.5
80 % NP-NP	177.8±1.4	129.4±3.6[b]	20.3±6.6	65.4±1.4	27.1±1.5
80 % NP-HP	182.5±3.0	127.9±4.8[b]	20.7±6.1	65.3±1.6	26.6±2.2
ad lib LP	181.4±4.5	140.8±1.1[a]	23.6±7.2	65.4±1.4	26.4±1.5
80 % LP-NP	176.0±3.9	128.3±5.6[b]	22.4±7.4	65.6±1.7	27.0±1.6
80 % LP-HP	181.5±9.1	121.7±4.2[b]	21.4±4.6	65.2±1.3	26.9±1.6
***p*-values**	0.529	0.004	0.494	0.944	0.696

Table 31 : Influence of feed restriction and protein content on protein and lipid content (g/kg whole bird inclusive feathers), abdominal fat content, carcase yield and breast meat percentage of Ross 508 broilers (42 days of age) (mean±SD)

	protein content	lipid content	abd. fat	yield (%)	breast meat (%)
ad lib NP	175.8±1.7 [a4]	135.0±18.8	22.5±6.5	65.9±1.7[bc]	27.1±1.8[ab]
80 % NP-NP	175.3 ±0.8[a]	131.8±15.8	21.1±4.9	65.3±1.9[ab]	27.2±1.7[ab]
80 % NP-HP	183.5 ±4.8[bc]	126.6±7.6	18.4±6.4	66.1±1.5[c]	27.6±1.8[ab]
ad lib LP	181.3±0.5[abc]	129.9±3.6	19.8±4.7	65.1±1.6[a]	26.8±2.2[a]
80 % LP-NP	177.3±4.9[ab]	139.5±2.2	21.6±6.2	65.9±1.6[bc]	28.1±1.3[b]
80 % LP-HP	187.3±7.5[c]	126.7±8.8	19.5±6.3	65.7±1.7[abc]	27.9±1.9[ab]
***p*-values**	0.026	0.707	0.511	0.075	0.171

4. Discussion

The influence of changing protein content in the diet of feed restricted broilers is quite different between both lines. This can be understood, as both lines are genetically very different. The initial retarded growth rate and especially the increased breast meat content of the 508-line are probably linked with the different reactions on protein content in the diet. Similar reactions are found in earlier work (Lippens and De Groote, 2000). The lowered protein content of the starter diet (- 2 %) too, can be considered as an early (qualitative) restriction. When comparing both *ad libitum* treatments ('ad lib NP' and 'ad lib LP'), no compensatory growth is found. Feed intake was not changed significantly and final body weights were lower for the birds on the low protein starter diet (differences not significant).

[4] means with the same letter are not significantly different from each other ($p<0.05$)

However, the growth retardation of the low protein fed birds was only minor or non-existing at 14 d of age (-3.4 % and +1.3 %, respectively for Ross 308 and Ross 508 respectively). It can be concluded that the reduction of the protein content in the starter was insufficient to reduce initial growth rate substantially.

When using a quantitative feed restriction, it seems recommendable to use a higher protein content in the grower. An easier catch-up growth in comparison with the control birds is found while FC ameliorates significantly. Especially for the 508-line some positive effects on carcase composition can be realised too. This is in agreement with the findings of Plavnik and Hurwitz (1989), who also found an increased need for almost all AA during the phase of compensatory growth. Jones and Farrell (1992a), however, supplemented the finisher diet with lysine and/or methionine and could not indicate consistent positive effects on zootechnical performances of feed restricted birds. This was also the case in a trial of Leeson and Zubair (1997) and Acar *et al.* (2001) in which only the lysine content of the diet was increased. In all these trials however, the possible positive effect of the increased AA contents can be confounded with the created imbalance between the AA. Indeed, with the supplementation of lysine and/or methionine, the ideal AA balance is disturbed. Strong deviations from this balance can induce a negative effect on growth performance (D'Mello and Lewis, 1970). In the study of Acar *et al.* (2001), a significantly increased *pectoralis minor* muscle yield, and not carcase yield, was found after lysine concentration was increased in the finisher diet of feed restricted birds. Positive effects on carcase composition of the increase of all AA were only found within the 508-line of the present trial.

Lowering the protein content of the starter did not change the technical performances of the broilers significantly in the present trial. This is in agreement with the results from Holsheimer *et al.* (1993) and Balbaie *et al.* (1999). However, a tendency for a worsened FC was found. A quantitative feed restriction prevented this deteriorating FC. This might be explained by the fact that a better N-utilisation and a reduced protein turn-over can be expected when birds are retarded in their initial growth and compensatory growth is induced (Lippens *et al.*, 2002b). On the other hand, attention should be paid to less favourable circumstances (e.g. low chick quality) in which case the combination of a low protein starter and feed restriction can result in additional minor performances.

No significant effects of dietary treatments on mortality were found. Indeed, as the data were from a rather small number of birds, the effect of restriction might have been obscured. More research with higher number of birds, comparable with the situation in practice, are probably necessary to be able to prove significant positive effects of feed restriction on mortality.

Chapter 8

INFLUENCE OF BROILER BREEDER AGE ON COMPENSATORY GROWTH CAPACITY

Adapted from :

Lippens, M. and Huyghebaert, G. (2003). Influence of broiler breeder age on compensatory growth capacity of their quantitative food restricted progeny broilers. British Poultry Science, submitted.

ABSTRACT

1. Two trials were carried out to investigate the influence of the age of broiler breeders on compensatory growth capacity of their progeny broilers (Ross 308 and Ross 508) after a quantitative feed restriction of 80 % of the *ad libitum*-intake starting d 4 for 4 d.
2. Three different ages of parent stock were used, namely young, middle age and old. These differences in breeder age induced some significant differences in one-day old chicken characteristics and probably in chicken quality.
3. Genetic background is one of the factors influencing compensatory growth capacity. Ross 508-chickens, which are already characterised by a relatively lower juvenile growth, seemed to have the highest ‘catch-up’-capacity.
4. The influence of breeder age on compensatory growth capacity was variable between the two trials. Chickens of a young parent stock showed a pronounced compensatory growth in the first trial, which was not confirmed in the second trial. There were indications that a more severe restriction might be needed for the chickens from the older parent stock.
5. It might be advised to control growth continuously in time by adjusting feed intake from day to day rather than using a constant quantitative feed restriction in all circumstances.

1. Introduction

Intensive research on quantitative feed restrictions is done at our Institute. A restriction to 80 % of the *ad libitum* intake from d 4 for 4 d seemed to be a good quantitative feed restriction to achieve the mentioned objectives. These preliminary studies however indicated variable results from trial to trial. In some trials final body weights and feed conversions of restricted birds were similar or significantly better in comparison with *ad libitum* fed animals. In other trials 'catch-up'-growth was insufficient to reach the weight of the control group. Also in literature, variable results of quantitative feed restrictions are found. This variability in literature has been attributed to a number of factors such as the nature, timing, severity and duration of the undernutrition but also genetic factors such as strain and sex. However these are insufficient to explain the differences found in our previous studies because these factors were eliminated as much as possible in the set-up of the trials.

In an attempt to find an explanation for the variable results, research was done looking at the influence of breeder age, as a possible indication for difference in chicken quality, on the compensatory growth capacity of their progeny broiler chickens. Indeed, a lot of factors influence one-day old chicken quality (incubation circumstances, age of the parent stock, transport conditions,…) and very often one-day old chicken quality is described in a rather subjective way. Age of the parent stock however, was chosen to be an easy way of creating a difference in one-day old chicken weight and mayby chicken quality. Indeed, a high correlation exists between age of the parent stock, egg-weight and day-old chick weight (McNaughton *et al.*, 1978; Shanawany, 1987; Sinclair *et al.*, 1990; Wilson, 1991; Peebles *et al.*, 1999a,b). Moreover, in the results of Boerjan (2002), it was stated that chicken quality (expressed in mean Pasgar©score) differs according to the age of the parent stock.

In literature, it has been found that there exists a correlation between day-old broiler weight and growth capacity (Morris *et al.*, 1968; Sinclair *et al.*, 1990). No research however has been published on the influence of day-old broiler weight (or chicken quality in general) on compensatory growth capacity of feed restricted birds. In other words, the question arises if day-old chicken weights, as an indication of chicken quality, are related with their 'catch-up' growth capacity when birds are feed restricted at an early age.

In the described study the effect of differences in age of the parent stock is investigated looking at performance, compensatory growth, mortality and carcase composition.

2. Materials and methods

2.1. Experimental design

Two multifactorial experiments (3 breeder ages x 2 lines x 2 feeding schedules) were set up to investigate the influence of breeder age on compensatory growth capacity of quantitative feed restricted broilers. There were 3 different ages of parent stock introduced, namely young (class 1), middle age (class 2) and old (class 3). The difference in ages of the parent stock, for both lines in the two trials, is described in Table 32. All chickens were hatched at our Institute in similar controlled conditions for both the incubator and the hatcher. Two lines of unsexed broiler chickens were used (Ross 308 and Ross 508). Sex ratios (males/females) at 42 days of age were 47/53, 52/48, 47/53, 46/54 for Ross 308, Ross 508 in trial 1 and trial 2, respectively. Correction for differences in sex ratios were carried out as mentioned in chapter 3. There were 2 feeding schedules either *ad libitum* or quantitatively restricted. Each treatment consisted of 4 replicates of 32 birds in trial 1 and 3 replicates of 32 birds in trial 2. Experiment 1 was conducted with 768 Ross 308 and 768 Ross 508 chicks. Experiment 2 was conducted with 576 Ross 308 and 576 Ross 508 chickens.

Table 32 : Age of the parent stock (weeks) of the different classes

	Trial 1		Trial 2	
	Ross 308	**Ross 508**	**Ross 308**	**Ross 508**
class 1	34	36	28	28
class 2	45	44	43	45
class 3	57	59	60	58

Mean daily feed consumption for each line and class was determined from the *ad libitum* groups and was assumed to be representative of the *ad libitum* intake. The quantitative feed restriction from d 4 to d 7 consisted of a feeding level to 80 % of the determined *ad libitum* intake of the previous 24 h.

2.2. Diets

The birds were fed *ad libitum*, except for the duration of the feed restrictions. A starter diet with 210 g CP/kg and 12.40 (12.42 in trial 2) MJ AME_n (broilers; CVB, 1997)/kg was given until 10 d of age. From d 11 until d 42 a grower diet with 196 g CP/kg (195 trial 2) and 12.85 MJ AME_n/kg was offered. The respective ingredient and nutrient composition is given in Table 33. As fishmeal was no longer allowed (BSE-legislation) just before the start of the second trial, new diets were formulated according to the least cost procedure. In this way also phytase was introduced in the diets.

2.3. Response parameters

General response parameters are mentioned in Chapter 3. At 43 d, 2 chickens per pen (1 male and 1 female) in trial 1 and 4 chickens per pen (2 males and 2 females) in trial 2 were used to determine the total lipid and protein content.

Table 33 : Ingredient composition and calculated nutrient composition of the diets (g/kg, unless otherwise stated)

	Trial 1		**Trial 2**	
Ingredients	**starter (0-10 d)**[1]	**grower (11-42 d)**[2]	**starter (0-10 d)**[1]	**grower (11-42 d)**[2]
Wheat	500.0	466.0	600.0	594.8
Soybeans (full fat)	155.0	200.0	156.2	189.6
Fish meal	46.2	50.0		
Soybean meal (48 % CP)	104.6	29.2	157.6	97.7
Soybean oil		0.6	2.3	
Yellow corn	115.8	130.0		29.7
Animal fat	40.0	39.4	37.7	47.0
Sorghum		50.5		
Dicalc. phosph. $2H_2O$	12.4	9.1	14.4	11.2
Limestone	8.05	7.55	9.20	8.80
Sodium chloride	1.48	1.82	2.35	2.41
Sodium bicarbonate	2.18	0.43	2.61	1.42
Vitamin/trace mineral mix	10.0	10.0	10.0	10.0
DL-methionine	1.91	1.97	2.19	2.21
L-lysine-HCl	1.53	2.67	4.05	3.85
L-threonine	0.70	0.63	0.89	0.75
Biofeed wheat	0.20	0.19	0.30	0.30
Ronozyme P			0.30	0.30
Nutrients (g/kg) (calculated)				
CP	210.0	195.9	210.0	195.0
AME_n, MJ/kg	12.40	12.85	12.42	12.85
$Isoleucine_{ad}$	7.9	7.2	7.9	7.2
$Leucine_{ad}$	13.5	12.7	12.8	11.9
$Lysine_{ad}$	10.8	10.7	12.2	11.1
$Meth._{ad}$ + $Cyst._{ad}$	7.9	7.5	7.9	7.5
$Phenyl._{ad}$ + $Tyr._{ad}$	14.1	12.9	14.3	13.1
$Threonine_{ad}$	7.2	6.5	7.2	6.5
$Tryptophan_{ad}$	2.1	1.9	2.1	2.0
$Valine_{ad}$	8.7	8.0	8.4	7.8
$Arginine_{ad}$	11.2	10.1	11.4	10.4
$Histidine_{ad}$	4.3	4.0	4.2	3.9
$NEAA_{ad}$	90.7	82.8	92.3	85.2

[1] containing 100 mg/kg monensin and 5 mg/kg flavomycine
[2] containing 1 mg/kg diclazuril and 5 mg/kg flavomycine (except for the last 5 d)

3. Results

3.1. Trial 1

3.1.1. One day old chicken characteristics

As to be expected eggs and 1-day old birds became heavier with increasing age of the parent stock (Table 34). Hatchability decreased with the age of the parent stock, especially for the Ross 508-chickens. Chicken to egg ratio however was not influenced by line or breeder age. On average, uniformity was higher for the Ross 308-chickens in comparison with the 508-line. A tendency for decreasing broiler uniformity (and increasing coefficient of variation) was found with advancing age of the parent stock.

Table 34 : Influence of line and parent stock age on quantitative measures of eggs and day-old chickens (mean ±SD) (Trial 1)

	Ross 308			Ross 508		
	class 1	class 2	class 3	class 1	class 2	class 3
egg weight	61.5±4.3	63.9±4.4	69.2±5.5	58.8±4.1	63.5±4.5	68.8±5.6
hatchability (%)	81.3	72.9	70.2	92.0	85.8	58.5
chicken weight	41.1±3.5	43.0±3.8	45.5±4.1	38.4±3.2	41.2±3.6	45.6±4.5
chicken/egg (%)	66.8	67.3	66.8	65.3	64.9	66.3
uniformity[3]	83	78	77	79	80	74
coefficient of variation (%)	8.8	8.7	9.0	8.4	8.7	9.8

3.1.2. Performance, mortality and uniformity

Results of Ross 308- and Ross 508-chickens for feed intake, body weight, gain, FC, mortality and uniformity at 42 d of age are shown in Table 35. In general, mean body weight gain and final body weight of feed restricted Ross 308-birds were significantly lower when compared to the birds fed *ad libitum*. However, a significant interaction indicates that this result should not be generalised over the three chicken qualities. Figure 28 shows that sufficient ‘catch-up’ growth was found for the chickens from the young parent stock (see later). Final body weight was 36 g higher when these birds were feed restricted. On the other hand, chickens from middle age and old parent stock had a 5 % lower final body weight with the induced feed restriction. In general, feed conversion was not significantly changed by feed restriction or chicken quality although a tendency for a better feed conversion (- 2.2 %) for the restricted 308-birds of the young parent stock was found (figures not shown).

[3] percentage weights between +/- 10 % of the mean

Total mortality (death and removed birds) tended to be lower for the restricted 308-birds in comparison with the controls. Breeder age however had no significant effect on mortality. A significant improvement of the uniformity with feed restriction was found for the 308-chickens. The effect of age of parent stock was not significant but in the line of 1st day-uniformity.

In contrast with the results for the 308-chickens, 508-chickens could compensate for the induced growth retardation (Table 35). The compensation in absolute figures, however, was only complete for the chickens of the young parent stock. Differences in final body weight between control and feed restricted birds were very limited for the chickens of the middle age parent stock (26 g) but rather high for chickens of the old parent stock (65 g). Also in contrast with the 308-chickens, the age of the parent stock had a significant effect on final body weight and weight gain of the 508-chickens. Chickens from an old parent stock kept the advantage of a high initial body weight until 42 d of age. They were significant heavier in comparison with the chickens of the two other classes. Their feed intake was higher, although differences were only significant between the birds from the young and the old parent stock.

Table 35 : Influence of feed restriction and broiler breeder age on performance of broiler chickens (42 days of age) (mean ±SD) (Trial 1)

ROSS 308	**feed intake (g/d)**	**body weight (g)**	**growth (g/b/d)**	**FC**	**total mortality (%)**	**uniformity**
treatment						
ad libitum	94.7±2.4[a4]	2312±64[a]	54.0±1.5[a]	1.754±0.049	3.8±2.7	63±8[a]
restricted	92.5±2.1[b]	2250±56[b]	52.5±1.4[b]	1.760±0.029	2.3±1.9	71±10[b]
class						
1	94.4±1.6	2281±56	53.3±1.3	1.772±0.047	3.7±2.5	72±8
2	93.3±3.5	2284±83	53.4±2.0	1.749±0.038	3.4±2.4	65±13
3	93.0±2.0	2278±67	53.2±1.6	1.750±0.034	2.0±2.4	63±5
ANOVA (*p*-values)						
treatment	0.026	0.009	0.010	0.702	0.105	0.042
class	0.435	0.973	0.944	0.435	0.313	0.139
interaction		0.015	0.016			
ROSS 508						
treatment						
ad libitum	90.7±2.9	2205±75	51.5±1.7	1.762±0.039	2.3±1.9	67±9
restricted	89.4±2.2	2175±59	50.8±1.4	1.759±0.030	2.0±3.3	67±9
class						
1	88.7±2.0[a]	2148±38[a]	50.2±0.9[a]	1.766±0.037	1.7±2.0	68±7
2	89.6±2.9[ab]	2176±56[a]	50.8±1.3[a]	1.762±0.041	1.4±2.0	65±11
3	91.9±1.8[b]	2247±66[b]	52.4±1.6[b]	1.753±0.026	3.4±3.5	67±10
ANOVA (*p*-values)						
treatment	0.176	0.197	0.205	0.861	0.839	1.000
class	0.039	0.006	0.009	0.784	0.305	0.849

[4] means with a different letter within a column and within each broiler line are significantly different from each other ($p<0.05$)

Again feed conversion was not significantly changed by restriction or breeder age. The positive effect of feed restriction on mortality found for the 308-chickens, could not be confirmed for the 508-chickens. Incidences of SDS, ascites and leg problems over the entire trial were too low to have any statistical or economical meaning. Treatment or class of the chickens did not change uniformity of the 508-birds.

3.1.3. Compensatory growth

A Gompertz function is used to estimate growth by treatment and by line during the whole growth trajectory in time. The functions and the corresponding R^2-values are shown in Figure 29. It confirms the pronounced 'catch-up'-growth of the restricted chickens of the young parent stock for both lines. Compensatory growth however was less pronounced for the Ross 508-chickens of the middle age parent stock. Compensatory growth was more or less absent in the 'older age'- groups.

3.1.4. Carcase composition

Table 36 shows a tendency for a lowered protein and lipid content in the carcase (whole bird

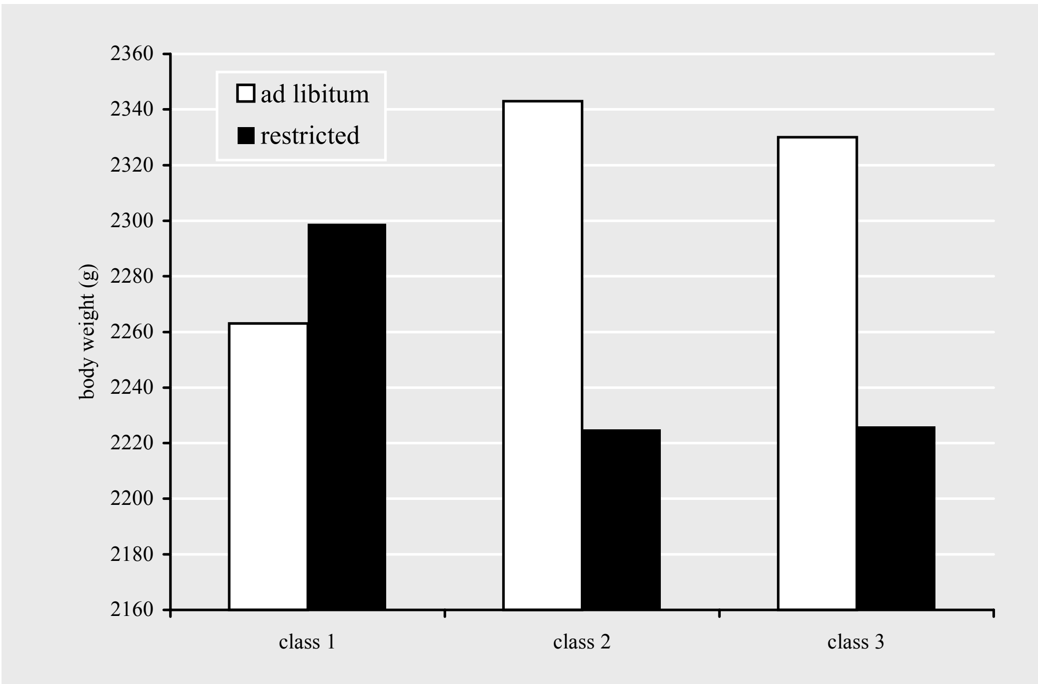

Figure 28 : Influence of breeder age and feed restriction on the final body weight of Ross 308 chickens at 42 d of age (Trial 1)

inclusive feathers) of feed restricted 308- and 508-birds. Abdominal fat was not influenced by feed restriction. The age of the parent stock had no major effect on all of these parameters. Also yield was not influenced by treatment or breeder age. However, a lower breast meat percentage was found after restricting the 308-chickens. When looking at the individual results, it becomes clear that this was only the case for chickens from middle age and old parent stock (no significant interaction).

Table 36 : Influence of feed restriction and age of the parent stock on protein and lipid content (g/kg whole bird inclusive feathers), abdominal fat content, carcase yield and breast meat percentage (42 days of age) (mean ±SD) (Trial 1)

ROSS 308	protein content	lipid content	abdominal fat (g/kg)	yield (%)	breast meat (%)
treatment					
ad libitum	183.0±5.4	142.6±7.7	21.7±1.8	66.0±1.5	27.4±0.9[a5]
restricted	180.0±2.3	136.5±7.8	21.2±2.1	65.8±0.9	26.7±0.7[b]
class					
1	181.9±2.8	143.6±8.5	22.4±1.6	65.3±1.6	27.1±0.7
2	180.7±4.7	136.7±10.3	20.9±1.9	66.1±1.1	27.3±1.1
3	182.0±5.5	138.2±3.6	20.9±2.0	66.4±0.8	26.8±0.9
ANOVA (*p*-values)					
treatment	0.117	0.065	0.530	0.719	0.039
class	0.810	0.187	0.210	0.260	0.509
ROSS 508					
treatment					
ad libitum	182.6±2.9	145.9±10.9	20.4±2.6	65.9±1.3	27.6±1.0
restricted	180.9±4.2	140.4±8.9	20.5±2.3	66.1±1.2	27.7±0.9
class					
1	180.9±3.5	142.5±7.8	20.5±2.5	66.3±0.8	27.8±1.2
2	182.5±4.1	146.3±9.8	19.1±2.3	65.6±1.4	27.6±0.8
3	182.0±3.5	140.8±12.6	21.6±2.0	66.1±1.3	27.6±0.9
ANOVA (*p*-values)					
treatment	0.275	0.214	0.909	0.652	0.660
class	0.692	0.571	0.144	0.520	0.916

3.2. Trial 2

3.2.1. One day old chicken characteristics

Again by using different ages of the parent stock, different weight classes for eggs and chickens were found. As an exception, chickens from the old 308-parent stock had the same mean body size as the birds of the middle age parent stock (Table 37). Chicken to egg ratio

[5] means with a different letter within a column and within each broiler line are significantly different from each other ($p<0.05$)

was not influenced by both parameters, but uniformity decreased with advancing age, with exception for the old parent stock of the 508-line.

3.2.2. Performance, mortality and uniformity

In this trial the body weight of feed restricted Ross 308-birds was not significant different from but somewhat lower in comparison with the *ad libitum* group (Table 38). Feed restriction also did not have any advantage for feed conversion. However, a trend of lowered mortality was found after feed restriction. The interaction however indicates that mortality was lowered only for the birds of the old parent stock. On the other hand, the age of the parent stock had no significant effect on performance.

Also for the Ross 508-chickens no difference in final body weight was found between feed restricted birds and *ad libitum* fed birds (Table 38). Moreover, the absolute values show an even higher final body weight after the juvenile growth retardation. Feed conversion was not significant lowered, but mortality was reduced with 50 %. A tendency for a better uniformity after restriction was only found for the 508-chickens.

Table 37 : Influence of line and parent stock age on quantitative measures of eggs and day-old chickens (mean ±SD) (Trial 2)

	Ross 308			**Ross 508**		
	class 1	class 2	class 3	class 1	class 2	class 3
egg weight	56.6±3.5	66.2±4.8	67.7±5.4	54.0±3.5	65.4±4.4	68.2±5.0
hatchability (%)	90.9	75.7	66.1	78.0	82.4	55.0
chicken weight	41.1±3.4	47.1±3.8	46.6±4.2	37.6±3.2	45.5±3.9	49.1±3.9
chicken/egg (%)	72.6	71.1	68.8	69.6	69.6	72.0
uniformity[6]	86 (8.3)	84 (8.2)	77 (9.0)	81 (8.4)	78 (8.5)	82 (8.0)
coefficient of variation (%)	8.3	8.2	9.0	8.4	8.5	8.0

In comparison with the results of trial 1, differences in 508-parent stock age again induced significant differences in performances. Performances at 42 d of age increased with advancing age of the parent stock. Mortality seemed to be negatively correlated with the quality of the chickens (chickens of a young parent stock have the highest mortality). Again cases of SDS and ascites over the total trial were very low (4 and 2 respectively).

[6] percentage weights between +/- 10 % of the mean

3.2.3. Compensatory growth

Figure 30 shows the Gompertz-curves (+ R^2-values) of the different treatments. In contrast with some quality classes of trial 1, there was no compensatory growth established by the Ross-308 chickens. Final body weight of restricted birds was lower in comparison with the *ad libitum*-group. The 508-chickens however seemed to have a higher capacity to catch up with the control growth. Mean final body weights of feed restricted birds coming from young or old parent stock were 1.7 % higher in comparison with the controls. This difference was even higher for the 508-chickens of the middle age parent stock, namely 3.4 % (figures not shown).

Table 38 : Influence of feed restriction and age of the parent stock on the performance of broiler chickens (42 days of age) (mean ±SD) (Trial 2)

ROSS 308	feed intake (g/d)	body weight (g)	growth (g/b/d)	FC	total mortality (%)	uniformity
treatment						
ad libitum	84.2±3.1	2067±71	48.1±1.7	1.750±0.033	8.4±6.8	58±10
restricted	83.2±3.5	2016±74	46.9±1.7	1.774±0.042	5.4±3.8	55±11
class						
1	82.1±3.7	2003±80	46.7±1.9	1.758±0.034	6.3±4.1	53±6
2	83.6±2.5	2036±79	47.4±1.9	1.767±0.033	5.0±4.0	60±10
3	85.4±3.0	2084±52	48.5±1.3	1.761±0.054	9.5±7.8	57±13
ANOVA (*p*-values)						
treatment	0.517	0.156	0.155	0.228	0.117	0.515
class	0.263	0.193	0.235	0.927	0.150	0.462
interaction					0.005	
ROSS 508						
treatment						
ad libitum	77.4±5.2	1855±109	43.1±2.5	1.795±0.06	14.5±.7.7^{a}	46±12
restricted	77.9±3.9	1897±125	44.1±2.9	1.768±0.06	7.0±5.7^{b}	56±10
class						
1	72.7±1.5^{a}[7]	1753±84^{a}	40.9±2.0^{a}	1.781±0.07	14.6±9.6^{a}	46±10
2	79.6±4.3^{b}	1895±68^{b}	44.0±1.6^{b}	1.809±0.07	11.3±6.9^{ab}	50±13
3	80.6±1.4^{b}	1980±48^{b}	46.0±1.1^{b}	1.754±0.03	6.4±4.1^{b}	58±7
ANOVA (*p*-values)						
treatment	0.717	0.233	0.234	0.399	*0.028*	0.056
class	*0.001*	*0.001*	*0.001*	0.383	0.121	0.152

[7] means with a different letter within a column and within each broiler line are significantly different from each other ($p<0.05$)

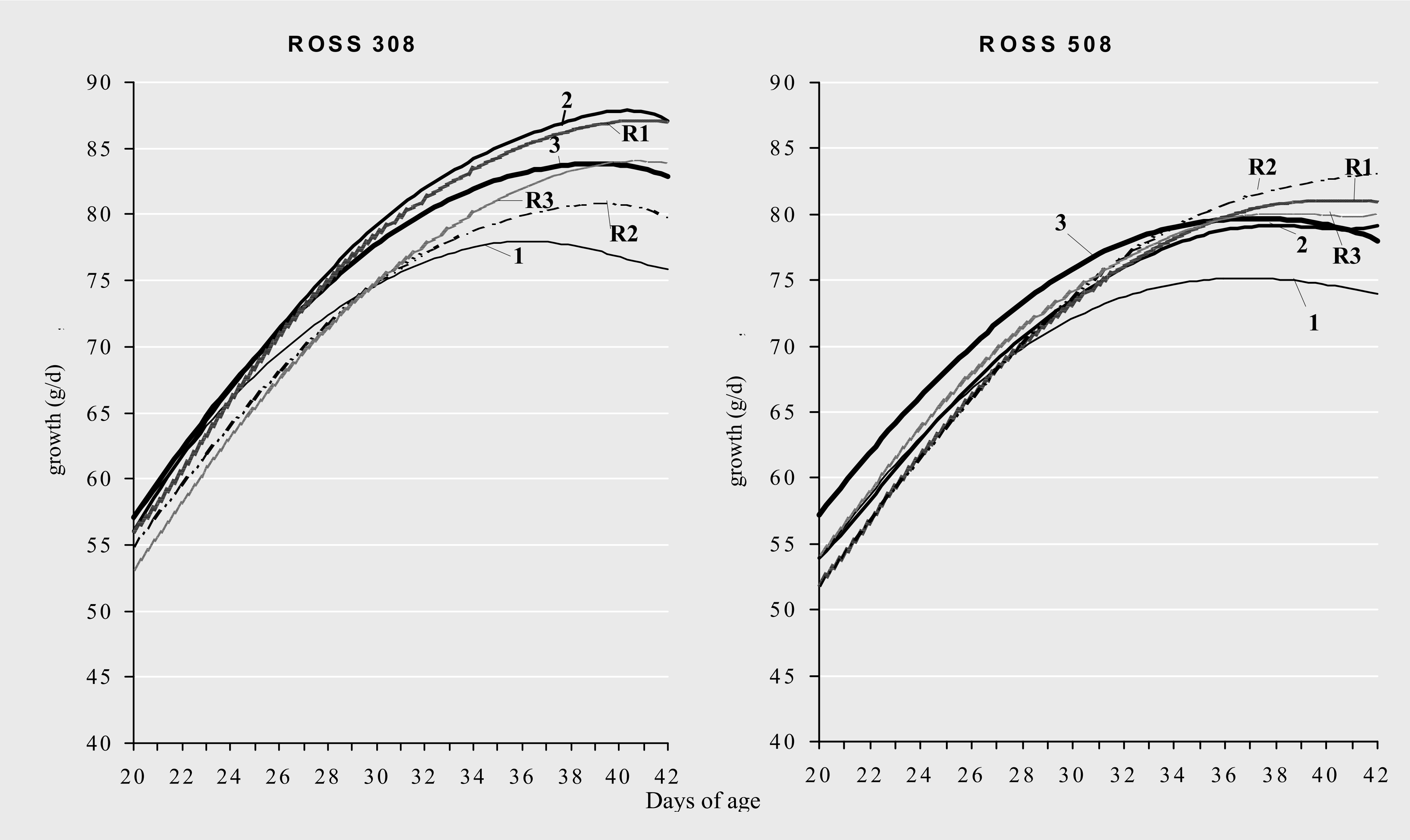

Figure 29 : Effect of feed restriction on growth from 20 to 42 d of age (Trial 1)

Ross 308-chickens

Ad lib – class 1 (**1**) : W_t=41.1 $e^{(0.20643/0.04319)(1-e^{-0.04319t})}$ R^2=0.999

Ad lib – class 2 (**2**) : W_t=43.0 $e^{(0.19457/0.03933)(1-e^{-0.03933t})}$ R^2=0.997

Ad lib – class 3 (**3**) : W_t=45.5 $e^{(0.19479/0.040446)(1-e^{-0.040446t})}$ R^2=0.998

Restricted – class 1 (**R1**): W_t=41.1 $e^{(0.19633/0.03941)(1-e^{-0.03941t})}$ R^2=0.998

Restricted – class 2 (**R2**) : W_t=43.0 $e^{(0.19549/0.04042)(1-e^{-0.04042t})}$ R^2=0.999

Restricted – class 3 (**R3**) : W_t=45.5 $e^{(0.18575/0.03804)(1-e^{-0.03804t})}$ R^2=0.998

Ross 508-chickens

Ad lib – class 1 (**1**) : W_t=38.4 $e^{(0.20659/0.04281)(1-e^{-0.04281t})}$ R^2=0.999

Ad lib – class 2 (**2**) : W_t=41.2 $e^{(0.19664/0.04043)(1-e^{-0.04043t})}$ R^2=0.997

Ad lib – class 3 (**3**) : W_t=45.6 $e^{(0.1982/0.04189)(1-e^{-0.04189t})}$ R^2=0.999

Restricted – class 1 (**R1**) : W_t=38.4 $e^{(0.19608/0.03931)(1-e^{-0.03931t})}$ R^2=0.999

Restricted – class 2 (**R2**) : W_t=41.2 $e^{(0.1900/0.03829)(1-e^{-0.03829t})}$ R^2=0.999

Restricted – class 3 (**R3**) : W_t=45.6 $e^{(0.19052/0.03977)(1-e^{-0.03977t})}$ R^2=0.998

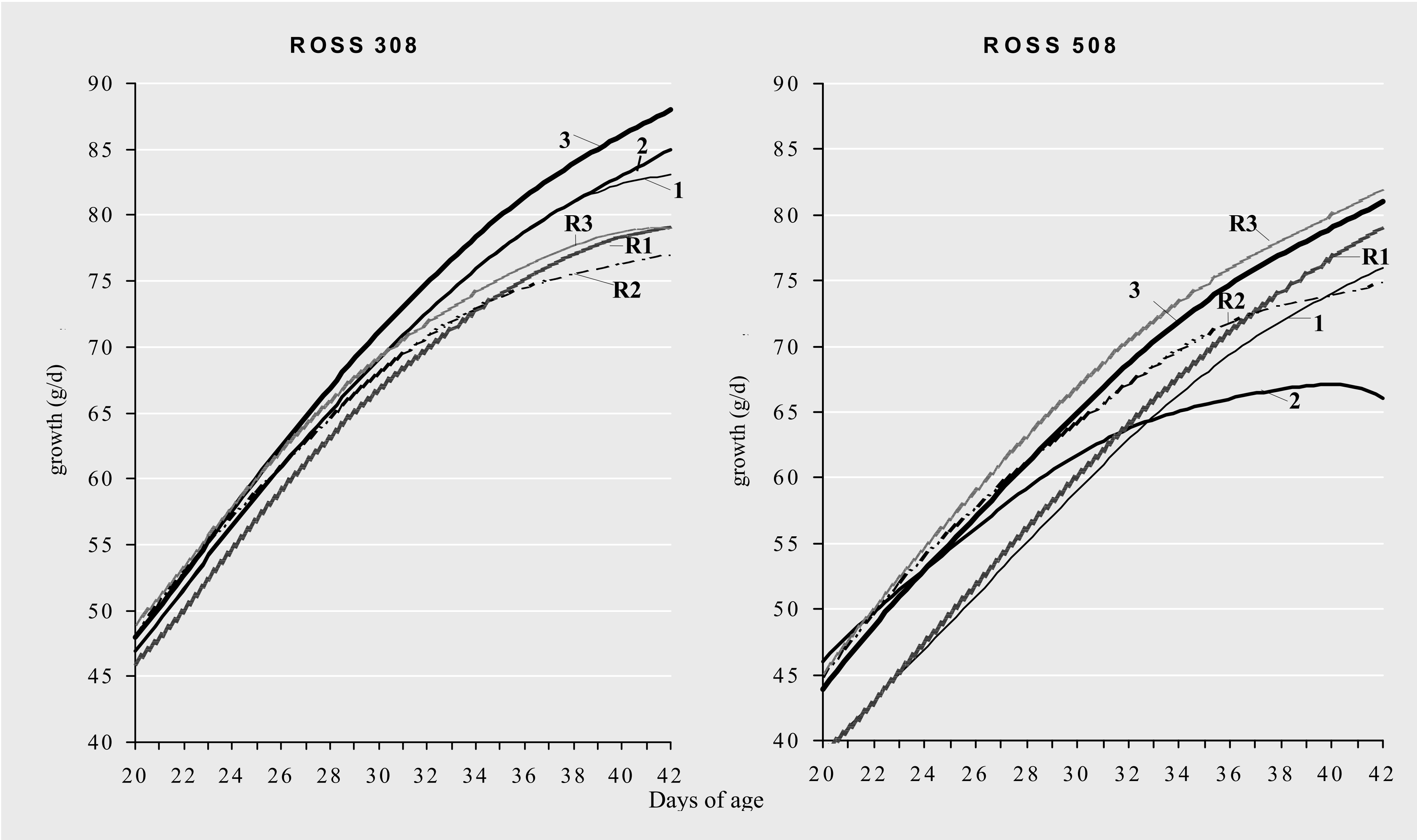

Figure 30 : Effect of feed restriction on growth from 20 to 42 d of age (Trial 2)

Ross 308-chickens

Ad lib – class 1 (**1**) : $W_t=41.1\ e^{(0.17844/0.035236)(1-e^{-0.035236t})}$ $R^2=0.997$

Ad lib – class 2 (**2**) : $W_t=47.1\ e^{(0.16809/0.03363)(1-e^{-0.03363t})}$ $R^2=0.995$

Ad lib – class 3 (**3**) : $W_t=46.6\ e^{(0.16972/0.03364)(1-e^{-0.03364t})}$ $R^2=0.999$

Restricted – class 1 (**R1**) : $W_t=41.1\ e^{(0.17727/0.03542)(1-e^{-0.03542t})}$ $R^2=0.997$

Restricted – class 2 (**R2**) : $W_t=47.1\ e^{(0.17681/0.03694)(1-e^{-0.03694t})}$ $R^2=0.993$

Restricted – class 3 (**R3**) : $W_t=46.6\ e^{(0.17646/0.03641)(1-e^{-0.03641t})}$ $R^2=0.997$

Ross 508-chickens

Ad lib – class 1 (**1**) : $W_t=37.6\ e^{(0.16646/0.03204)(1-e^{-0.03204t})}$ $R^2=0.996$

Ad lib – class 2 (**2**) : $W_t=45.5\ e^{(0.17923/0.03866)(1-e^{-0.03866t})}$ $R^2=0.992$

Ad lib – class 3 (**3**) : $W_t=49.1\ e^{(0.16132/0.03260)(1-e^{-0.03260t})}$ $R^2=0.999$

Restricted – class 1 (**R1**) : $W_t=37.6\ e^{(0.16504/0.03130)(1-e^{-0.03130t})}$ $R^2=0.992$

Restricted – class 2 (**R2**) : $W_t=45.5\ e^{(0.17233/0.035655)(1-e^{-0.035655t})}$ $R^2=0.995$

Restricted – class 3 (**R3**) : $W_t=49.1\ e^{(0.16392/0.03323)(1-e^{-0.03323t})}$ $R^2=0.996$

3.2.4. Carcase composition

No effects of feed restriction or parent stock age are found on protein and lipid content (Table 39). Only for the 508-chickens there seemed to be a significant lower abdominal lipid content in chickens from a young parent stock in comparison with chickens from the other two quality classes. No negative effects on yield and breast meat percentage should be expected when the birds of the different quality classes are restricted.

Table 39 : Influence of feed restriction and age of the parent stock on protein and lipid content (g/kg whole bird inclusive feathers), abdominal fat content, carcase yield and breast meat percentage (42 days of age) (mean ±SD) (Trial 2)

ROSS 308	protein content	lipid content	abdominal fat (g/kg)	yield (%)	breast meat (%)
treatment					
ad libitum	178.9±3.9	128.2±8.4	18.2±2.5	66.5±1.2	26.6±1.2
restricted	177.5±3.5	131.2±6.3	17.6±2.1	66.5±1.0	26.8±0.9
class					
1	179.2±3.1	132.0±5.9	16.8±2.4	65.9±1.6	26.8±1.4
2	176.3±3.8	132.5±8.2	18.5±2.3	66.6±0.7	26.7±0.9
3	179.0±4.0	124.6±6.0	18.6±2.0	67.0±0.5	26.6±1.1
ANOVA (*p*-values)					
treatment	0.473	0.372	0.609	0.986	0.628
class	0.402	0.121	0.406	0.270	0.958
ROSS 508					
treatment					
ad libitum	175.5±3.3	131.8±10.5	17.9±2.7	66.5±1.0	27.6±1.4
restricted	177.6±3.4	132.7±7.8	18.8±3.6	67.0±1.0	28.0±0.8
class					
1	175.1±1.1	126.2±9.5^{a}[8]	15.5±1.8^{a}	66.6±1.1	28.1±0.8
2	175.8±3.7	131.9±8.3^{ab}	20.0±2.5^{b}	66.5±1.2	27.3±1.6
3	178.8±4.0	138.8±4.1^{b}	19.6±3.0^{b}	67.0±0.8	27.9±1.6
ANOVA (*p*-values)					
treatment	0.207	0.819	0.459	0.339	0.497
class	0.165	0.060	0.018	0.706	0.531

4. Discussion

According to Tona *et al.* (2001), eggs produced by young or old breeders do not hatch as well as those from breeders in between. The high hatchability of the eggs (% of total eggs setted) from the young parent stock in the present trial does not agree with these findings. In the case of trial 1, however, the age of the young breeders was not as low as mentioned in the publication of Tona *et al.* (2001). The lower hatchability of eggs of the older parent stock

[8] means with a different letter within a column and within each broiler line are significantly different from each other ($p<0.05$)

might be due to both thinner, more porous shells (increased breakage and contamination) but mainly the lower fertility (Wilson, 1991). Indeed, in broiler breeder husbandry, fertility is negatively affected by too heavy body weights of females and mainly of males, resulting in worse mating behaviour.

With increasing age of the parent stock, higher egg and day-old chick weights are found which is in agreement with findings in literature (McNaughton *et al.*, 1978; Shanawany, 1987; Sinclair *et al.*, 1990; Wilson, 1991; Peebles *et al.*, 1999a,b). In other words, it might be expected that the present differences in breeder age created some differences in one-day old chicken quality.

In literature there are some conflicting results on the effect of differences in age of the parent stock on the final body weights of the broilers. The general statement is that larger hatching eggs result in larger 1-day old chickens, which in turn results in heavier broilers at market age. Indeed, in the study of Sinclair *et al.* (1990) e.g., using two sets of breeders of 30 and 50 wk of age respectively, there was a strong positive correlation between breeder age and final body weight at 6 weeks of age. Peebles *et al.* (1999a) demonstrated that broilers from hens of 51 wk of age performed however better than those from 63-wk-old hens which in turn were significantly better than those from 35-wk-old breeders. Also in the present trials, it was not always possible to find a straight-line effect of different breeder ages on the growth rate or final body weight of the broilers. Results in literature on the effect of breeder age on feed conversion is even more variable with no clear conclusion (Wilson, 1991). In the present trial no significant differences were found.

One of the factors influencing growth and probably compensatory growth capacity is the genetic background (Scheideler and Baughman, 1993; Zubair and Leeson, 1996a; Lippens *et al.*, 2000). Indeed in the current trial, it seems that Ross 508-chickens, which are already characterised by a relatively low juvenile growth rate, have the highest capacity to establish compensatory growth after a feed restriction. This is in agreement with some earlier findings (Lippens, 2001; Lippens *et al.*, 2002b; Lippens *et al.*, 2002c) and rejects the hypothesis mentioned in chapter 4 (Lippens *et al.*, 2000) that the low juvenile growth rate of 508-birds limits their compensatory growth capacity. On the contrary, it is possible that the genetic-linked growth rate of the 508-line (versus the 308-line) might be an essential condition for a more complete catch-up growth after a quantitative feed restriction. This corresponds with the statement of Cherry *et al.* (1978) that slower growing birds exhibit a higher compensatory growth rate after restriction in comparison with faster growing strains.

Next to genetics, also the age of the parent stock has some influence on compensatory growth capacity although with changing effects. Very remarkable was the pronounced 'catch-up'-growth of the chickens of the young parent stock in trial 1. One would however expect that these chickens, with their low starter weights, could not tolerate any restriction. This response could, however, not be confirmed in the second trial. Very remarkable is the difference in growth rate of the control groups during the last week. In the first trial, control birds reached a maximum in growth very early, while no maximum was found in the second trial. This might be linked with the level of the general performances of the birds, which was higher in trial 1 in comparison with trial 2.

Both protein and energy contents were constant in the diets of both trials, however, due to least cost formulation, some changes in amino acid ratios (relative to lysine) were found. Based on the findings of Lippens *et al.* (1997), there are no indications that the increased lysine concentration in trial 2 could have had a negative effect on the zootechnical performances. Moreover, the same diet gave good results in a trial described in the next chapter.

On the other hand, differences in results between the successive trials might be correlated with differences in health and immune status. Chicken quality however is also affected by incubation circumstances. Within each trial of the present research the chicken to egg ratio (%) is not significantly influenced by age of the parent stock. The pronounced difference of this ratio between the two trials (66 % and 71 %, respectively) however may indicate a difference in chicken quality in time. Indeed, according to Deeming (2000), values between 65-68 % indicate a good quality. Values above 70 % are worse, namely eggs usually incubated under too high humidity conditions. In other words, this might explain partly differences in mean growth figures in the two trials and as a consequence difference in compensatory growth capacity. The absence of any compensatory growth for the chickens of an older parent stock on the other hand could indicate the need for a more severe feed restriction in these circumstances.

As this variability in growth was found, some differences in carcase composition would also be expected. These were only minor in the present research. However, final weight of restricted birds should not deviate too much from their respective controls to avoid obvious reductions of breast meat percentage.

Despite the lack of consistent improvement of the performances, using quantitative feed restrictions may still have some advantage. Indeed, possibilities to reduce mortality were found. Although few mean mortality rates were significantly different, they can be of very economic importance. As mentioned in Lippens *et al.* (2000), the lack of significance may have to do with the relatively low number of animals used in the trials. The positive tendency for a better uniformity might give a supplemental advantage.

In general, there seemed to be no correlation between day-old chicken weight and compensatory growth capacity. As the results were not consistent between trials, it can be concluded that it is probably not useful to introduce the identical quantitative feed restriction schedules in all circumstances.

Chapter 9

INFLUENCE OF FEED STRUCTURE ON COMPENSATORY GROWTH CAPACITY

Adapted from :

Lippens, M. and Huyghebaert, G. (2003). Influence of food structure on compensatory growth capacity and carcase composition of two quantitative food restricted broiler lines. British Poultry Science, submitted.

ABSTRACT

1. Two experiments were conducted each with two Ross broiler lines (Ross 308 and Ross 508) to evaluate the possible difference in compensatory growth capacity after a quantitative feed restriction using either mash or pellets. The effect on performance and carcase composition was examined.
2. The quantitative feed restriction started from d 4 until d 7 and consisted of a feed supply to 80 % of the determined *ad libitum* intake of the previous 24 h. After the period of restriction, all birds were fed *ad libitum*.
3. In both trials, and for both lines, restrictions did not influence final body weight and FC significantly. On the other hand, pelleting feeds gave higher final body weight and better feed conversion in comparison with mash feeding.
4. Pellet feeding did not increase mortality significantly. Feed restriction, however, induced a trend for a lower mortality. There were no indications that this positive effect depended on feed texture.
5. Compensatory growth did not occur when using pellet feeds. In contrast, when using mash feeding, compensatory growth was found, although not in all cases. Also the age at which compensatory growth started was variable.
6. No consistent effects of feed restriction on carcase composition were found. On the other hand, pellets had a rather positive effect on yield and breast meat percentage.
7. Many factors can play a role in the ability of the bird to realise compensatory growth after early, temporary feed restriction. The variation found can be partially explained by feed structure. In general, a mild feed reduction at an early age can give some economical advantages, mainly by lowering mortality. However, attention should be paid to carcase yield and breast meat percentage, especially when feed restriction is combined with pelleted feeds or compensatory growth is insufficient to compensate completely for early growth retardation.

1. Introduction

Genetic selection and optimisation of nutrition have contributed to increasing growth rates and higher body weights at market age of modern broiler lines. These increased growth rates are associated with a higher feed and nutrient intake. On the other hand, they have also led to a more frequent occurrence of metabolic diseases, skeletal disorders and increased fat deposition.

It is well established that crumbles and pellets give higher growth rates than mash diets (Savory, 1974; Wenk and Van Es, 1979; Choi *et al.*, 1986; Proudfoot and Hulan, 1989; Hamilton and Proudfoot, 1995; Urdaneta-Rincon and Leeson, 2002). According to Nir (1997) adaptation to changes in feed structure includes feed consumption, capacity and evacuation of the gastro-intestinal tract, synthesis and secretion of digestive enzymes, lipogenesis, eating behaviour and activity. Many studies have shown that birds fed pellets, spend less time eating and more time sitting in comparison with meal feeding thereby influencing energy conversion (Jensen *et al.*, 1962; Savory, 1974; Nir *et al.*, 1994; Nir, 1997). Also, due to the pelleting process, a higher digestibility of some nutrients or inactivation of heat-labile toxic factors can be realised, depending on feed ingredients used (Calet, 1965; Summers *et al.*, 1968; Huyghebaert and De Groote, 1979, 1980; Wenk and Van Es, 1979; Leeson and Summers, 2001). In general, it is to be expected that with these very high growth rates the occurrence of the mentioned metabolic diseases will be more pronounced (Proudfoot *et al.*, 1982; Proudfoot and Hulan, 1989).

To avoid these negative effects, feed restriction programmes have been introduced. If growth is restricted early in life, a better development of the vital organs becomes possible. Indeed, as mentioned before, early feed restriction causes a shift in nutrient and energy supply in favour of the early maturing supply organs rather than the demanding tissues like muscle (Govaerts *et al.*, 2000). As predisposition for metabolic disorders already occurs during the first weeks of life, it can be understood that reducing initial growth and thus, the metabolic load and oxygen requirements in this crucial phase, is a good way of avoiding metabolic disorders (Buys et al., 1998). The consequent compensatory growth is expected to give similar final body weights and better feed conversion in comparison with *ad libitum* fed birds (Plavnik and Hurwitz, 1985, 1988, 1991). Moreover, these programmes are able to reduce leg problems and mortality (Robinson *et al.*, 1992; Saleh *et al.*, 1996; Carter *et al.*, 1994; Lippens *et al.*, 2000). Metabolic diseases can be diminished (Arce *et al.*, 1992; Gonzales *et al.*, 1998; McGovern *et al.*, 1999; Lippens *et al.*, 2000; Urdaneta-Rincon and Leeson, 2002). However, results in the literature relating to restriction programmes are rather variable. Indeed, a lot of factors such as nature, timing, severity and duration of the restriction or genetic factors influencing the compensatory growth capacity are well documented (Yu and Robinson, 1992; Zubair and Leeson, 1996a; Lippens *et al.*, 2000; Lippens and Huyghebaert, 2003). An additional factor of influence might be the feed structure. As feed structure has a pronounced effect on growth rate and feed conversion, it might have a significant effect on the capacity of the birds to compensate for early feed restriction. Most of the results in the literature concern pellets. However, meal diets are common practice in Belgium. The question arises whether or not a certain feed restriction on either meal or pellets would have similar effects on the entire growth curve.

Moreover, some results in the literature show that, when using restriction programmes, losses in carcase yield and breast meat yield should be expected (Scheideler and Baughman, 1993; Van Harn and Fabri, 1995; Van Harn en Van Middelkoop, 1996; 1998; Urdaneta-Rincon and Leeson, 2002). However in our research, there was no indication of these losses when compensatory growth was nearly complete (Lippens *et al.*, 2000; Lippens, 2001; Lippens and Huyghebaert, 2003). This confirmed earlier findings of Leeson *et al.* (1991); Van Harn (1992), Zubair and Leeson (1994a); Carter *et al.* (1994) and Palo *et al.* (1995). Part of this variation might be explained by feed structure. No studies are known where quantitative feed restriction combined with either pellets or mash feeding in the same trial examined the effect on carcase composition.

The purpose of this research was to evaluate the impact of feed texture ‘pellets and mash’ on compensatory growth capacity after an early quantitative feed restriction. Zootechnical performance and carcase composition were used as parameters.

2. Materials and methods

2.1. Experimental design

Two multifactorial experiments (2 lines x 2 feeding schedules x mash/pellet) were set up to investigate the influence of feed structure on compensatory growth capacity of quantitatively feed restricted broilers.

Two lines of unsexed broiler chickens were used (Ross 308 and Ross 508). Both experiments were conducted with 384 broilers of each line with 3 replicates of 32 animals per treatment. All chickens were hatched in equally controlled conditions in the hatchery of the Institute.

The ages of the parent stock in trial 1 were 46 and 44 weeks for Ross 308 and Ross 508, respectively. For trial 2, both parent stocks aged 43 weeks of age. In trial 1, mean day-old weights were 45.9 g (8.3 % coefficient of variation) and 42.2 g (8.1 % coefficient of variation) for Ross 308- and 508-birds, respectively. In the second trial, this was 44.1 g (8.6 %) and 41.9 g (8.6 %), respectively. The progeny broilers were either fed *ad libitum* or quantitatively restricted and feeds were in mash or pellet form (during the entire trial). The quantitative feed restriction from d 4 to d 7 consisted of a feed supply of 80 % of the determined *ad libitum* intake of the previous 24 h.

2.2. Diets

The birds were fed *ad libitum*, except for the duration of the feed restriction. A starter diet with 210 g CP/kg and 12.40 MJ AME_n (broilers; CVB, 1997)/kg was given until 10 d of age. From 11 d until 42 d a grower diet with 195 g CP/kg and 12.85 MJ AME_n/kg was offered. The respective ingredient and nutrient compositions are given in Table 40. As fishmeal was no longer allowed (BSE-legislation) just before the start of the second trial, new diets were formulated according to the least cost procedure. In this way also phytase was introduced in the diets. Mash feeds were pelleted (2.8 mm diameter) in our own feed mill. Pellet quality is recorded in Table 41.

2.3. Response parameters

General response parameters are mentioned in Chapter 3. At 43 d four chickens per pen (2 males and 2 females) were used to determine total lipid and protein content.

3. Results

3.1. Performance, mortality and uniformity

Body weights at 8 d of age were significant lower for the restricted birds in both trials and for both lines (trial 1-Ross 308 : 145 g, 122 g, 175 g, 152 g; trial 1-Ross 508 : 148 g, 129 g, 168 g, 147 g; trial 2-Ross 308 : 148 g, 135 g, 168 g, 149 g; trial 2-Ross 508 : 145 g, 128 g, 170 g, 142 g; for meal *ad lib*, meal restricted, pellets *ad lib* and pellets restricted, respectively). In both trials, and for both lines, catch up growth after feed restriction was more or less complete

(Tables 42 and 43). The mean weight of the restricted broilers was not significantly different from that of their *ad libitum* counterparts. In most cases, however, birds fed *ad libitum* were numerically heavier. Only for the Ross 308-birds of the second trial mean final body weight of the restricted birds (mean mash+pellets) was, in absolute values, higher than the controls (Table 43). In all cases, the overall feed conversion was not changed significantly by feed restriction.

Table 40 : Ingredient composition and calculated nutrient composition of the diets (g/kg, unless otherwise stated)

	Trial 1		**Trial 2**	
Ingredients	**starter (0-10d)[1]**	**grower (11-42 d)[2]**	**starter (0-10 d)[1]**	**grower (11-42 d)[2]**
Wheat	500.0	466.0	600.0	594.8
Soybeans (full fat)	155.0	200.0	156.2	189.6
Fish meal	46.2	50.0		
Soybean meal (48 % CP)	104.6	29.2	157.6	97.7
Soybean oil		0.6	2.3	
Yellow corn	115.8	130.0		29.7
Animal fat	40.0	39.4	37.7	47.0
Sorghum		50.5		
Dicalc. phosph. $2H_2O$	12.4	9.1	14.4	11.2
Limestone	8.05	7.55	9.20	8.8
Sodium chloride	1.48	1.82	2.35	2.41
Sodium bicarbonate	2.18	0.43	2.61	1.42
Vitamin/trace mineral mix	10.0	10.0	10.0	10.0
DL-methionine	1.91	1.97	2.19	2.21
L-lysine-HCl	1.53	2.67	4.05	3.85
L-threonine	0.70	0.63	0.89	0.75
Biofeed wheat	0.20	0.19	0.30	0.30
Ronozyme P			0.30	0.30
Nutrients (g/kg) (calculated)				
CP	210	195	210	195
AME_n, MJ/kg	12.40	12.85	12.40	12.85
$Isoleucine_{ad}$	7.9	7.2	7.9	7.2
$Leucine_{ad}$	13.5	12.7	12.8	11.9
$Lysine_{ad}$	10.8	10.7	12.2	11.1
$Meth._{ad}$ + $Cyst._{ad}$	7.9	7.5	7.9	7.5
$Phenyl._{ad}$ + $Tyr._{ad}$	14.1	12.9	14.3	13.1
$Threonine_{ad}$	7.2	6.5	7.2	6.5
$Tryptophan_{ad}$	2.1	1.9	2.1	2.0
$Valine_{ad}$	8.7	8.0	8.4	7.8
$Arginine_{ad}$	11.2	10.1	11.4	10.4
$Histidine_{ad}$	4.3	4.0	4.2	3.9
$NEAA_{ad}$	90.7	82.8	92.3	85.2
Ca	9.0	8.0	9.0	8.0
P_{av}	4.6	4.0	4.6	4.0
Cl	2.3	2.8	2.5	2.5
Na	1.8	1.5	1.8	1.5

[1] containing 100 mg/kg monensin and 5 mg/kg flavomycine
[2] containing 1 mg/kg diclazuril and 5 mg/kg flavomycine (except for the last 5 d)

Table 41 : Pellet quality of the diets

	%			
	Trial 1		Trial 2	
sieve mm	**starter (0-10d)**	**grower (11-42 d)**	**starter (0-10 d)**	**grower (11-42 d)**
<0.125	0.0	0.0	0.0	0.0
0.125	0.0	0.1	0.0	0.0
0.25	0.9	1.6	1.0	2.9
0.5	1.9	1.8	1.5	5.3
1	3.4	2.2	2.8	6.1
2	92.8	93.2	94.3	85.4
4	0.8	0.6	0.4	0.3

Table 42 : Influence of feed restriction and feed structure on performance of broiler chickens (42 days of age) (mean±SD) (Trial 1)

ROSS 308	**feed intake (g/d)**	**body weight (g)**	**growth (g/b/d)**	**FC**	**total mortality (%)**	**uniformity**
treatment						
ad libitum	91.9±2.2[a3]	2147±65	50.0±1.6	1.837±0.049	9.1±3.3	56.7±8.0
restricted	89.0±4.1[b]	2092±120	48.7±2.9	1.829±0.050	5.9±2.7	52.7±10.9
feed						
mash	88.7±4.1[a]	2059±98[a]	47.9±2.3[a]	1.852±0.061	7.3±3.4	52.2±9.8
pellets	92.2±1.3[b]	2181±45[b]	50.8±1.1[b]	1.814±0.020	7.7±3.6	57.2±9.1
ANOVA (*p*-values)						
treatment	0.024	0.173	0.171	0.785	0.579	0.487
feed	0.009	0.011	0.011	0.228	0.406	0.389
interaction	0.007					
ROSS 508						
treatment						
ad libitum	86.3±2.9	2070±65	48.3±1.5	1.788±0.030	5.0±4.0	63.0±6.6[a]
restricted	84.9±4.2	2005±117	46.7±2.8	1.818±0.063	3.6±3.3	49.5±11.6[b]
feed						
mash	83.2±3.0[a]	1979±102[a]	46.1±2.4[a]	1.807±0.067	5.9±4.0	51.5±13.2[a]
pellets	88.0±2.2[b]	2096±42[b]	48.9±1.0[b]	1.798±0.030	2.7±2.4	61.0±7.7[b]
ANOVA (*p*-values)						
treatment	0.338	0.172	0.170	0.366	0.088	0.038
feed	0.010	0.027	0.026	0.795	0.203	0.008
interaction						0.038

In relation to feed structure however, the results show that pellets had a significant positive effect on the final body weight. In most cases, feed intake was increased when using pellets. Differences were, however, only significant in trial 1. Moreover the increases due to pelleting feed were more pronounced for feed restricted broilers (significant interaction Ross 308-broilers in trial 1). Using pellets also enhanced feed conversion. However, only in the second trial the differences were significant.

[3] means with a different letter within a column and within each broiler line are significantly different from each other ($p<0.05$)

Table 43 : Influence of feed restriction and feed structure on performance of broiler chickens (42 days of age) (mean±SD) (Trial 2)

ROSS 308	feed intake (g/d)	body weight (g)	growth (g/b/d)	FC	total mortality (%)	uniformity
treatment						
ad libitum	90.3±5.9	2289±166	53.5±4.0	1.691±0.054	9.4±9.4	71.7±16.3
restricted	91.3±3.9	2311±114	54.0±2.7	1.693±0.048	7.0±7.4	73.5±8.7
feed						
mash	89.0±4.9	2203±110[a][4]	51.4±2.6[a]	1.731±0.025[a]	3.6±3.1	70.2±9.0
pellets	92.6±4.3	2398±78[b]	56.1±1.8[b]	1.652±0.028[b]	12.8±9.3	75.0±15.7
ANOVA (*p*-values)						
treatment	0.737	0.720	0.719	0.921	0.596	0.828
feed	0.244	0.011	0.011	0.002	0.071	0.571
ROSS 508						
treatment						
ad libitum	88.5±2.6	2274±111	53.1±2.7	1.666±0.048	10.9±4.8	67.7±10.4
restricted	87.4±2.0	2213±82	51.7±1.9	1.692±0.037	5.2±5.1	65.8±6.3
feed						
mash	87.5±1.5	2186±48[a]	51.1±1.1[a]	1.714±0.022[a]	9.3±6.6	62.7±9.3
pellets	88.4±2.9	2300±106[b]	53.8±53.8[b]	1.644±0.026[b]	6.8±4.6	70.8±4.8
ANOVA (*p*-values)						
treatment	0.509	0.245	0.248	0.070	0.102	0.704
feed	0.565	0.047	0.045	0.001	0.434	0.117

Mortality levels were variable and none of the effects were significant in either trial, although in trial 2 values for Ross 308 were numerically higher with pellet feeding. Feed restriction tended to lower losses due to mortality and culling. For the Ross 508-chickens probability values were 0.09 and 0.10 for trial 1 and 2 respectively.

Meal fed Ross 508-chickens in trial 1 showed a lower uniformity after feed restriction (significant interaction). However, this was not confirmed in the other trial results.

3.2. Compensatory growth

Figure 31 and 32 show the growth curves (based on Gompertz equations) of the different lines and the different treatments. It is clear from these results that compensatory growth, induced by the same feed restriction, varied by trial, by feed structure and by line.

The relative reduction in body weight shortly after restriction was similar for both mash and pellet feeding (approximately 20 g). However, using pelleted feeds, it seems that this restriction was not sufficient to induce any substantial compensatory growth (Figures 31 and 32). Final body weights remained lower in comparison with the control group (except for Ross 308 in trial 1). Differences were not significant.

[4] means with a different letter within a column and within each broiler line are significantly different from each other ($p<0.05$)

In contrast, when using mash feeding, compensatory growth was found, although not in all circumstances. Also the age of initiating compensatory growth was variable. In the first trial Ross 308 chickens realised no compensatory growth resulting in significant lower final body weights (2123 g and 1994 g, respectively). Ross 508-chickens in this trial only showed a compensatory growth during the last week. This was however insufficient to reach the target of the controls (2028 g and 1930 g, respectively). In trial 2 on the other hand, the restricted Ross 308-birds grew already faster than the controls immediately after the restriction. This explains the higher final body weights of the restricted birds (2171 g and 2235 g, respectively). Ross 508-chickens did not realise any compensatory growth (2213 g and 2160 g, respectively).

When comparing mash with pellet feeding, it is clear that mash feeding also is a kind of feed restriction. Indeed, when comparing the *ad libitum*-groups of both feed structures, growth retardation was in most cases followed by 'compensatory growth'. As mentioned before, this was not sufficient to catch up with the weights of the pellet-fed birds.

Table 44 : Influence of feed restriction and feed structure on protein and lipid content (g/kg whole bird inclusive feathers), abdominal fat content, carcase yield and breast meat percentage (42 days of age) (mean±SD) (Trial 1)

ROSS 308	**protein content**	**lipid content**	**abd. fat (g/kg)**	**yield (%)**	**breast meat (%)**
treatment					
ad libitum	182.9±1.8(n=5)[5]	124.2±2.8(n=5)	17.6±2.7	68.2±1.9	27.0±0.8
restricted	178.8±2.7(n=3)	118.9±3.8(n=3)	19.4±4.0	68.4±1.4	26.6±1.4
feed					
mash	181.9±1.9(n=4)	120.2±5.7(n=4)	20.3±3.4	67.0±1.5	26.5±1.5
pellets	179.8±3.6(n=4)	122.8±2.5(n=4)	16.7±2.4	69.6±6.4	27.1±6.4
ANOVA (*p*-values)					
treatment	0.061	0.069	0.328	0.882	0.531
feed	0.275	0.292	0.081	0.121	0.414
ROSS 508					
treatment					
ad libitum	185.6±5.4(n=4)	115.9±7.2	15.9±2.1	69.1±2.2	28.3±7.3[a6]
restricted	182.8±3.3(n=4)	115.7±5.1	16.8±2.5	67.8±1.4	27.5±9.6[b]
feed					
mash	184.7±4.6(n=5)	113.9±6.8	16.5±2.5	68.7±2.4	27.3±8.8[a]
pellets	183.7±4.3(n=3)	117.6±3.1	16.2±2.2	68.2±1.5	28.5±5.0[b]
ANOVA (*p*-values)					
treatment	0.483	0.966	0.522	0.297	0.043
feed	0.783	0.521	0.805	0.681	0.009

[5] due to a technical problem, some replicates were lost. The number of replicates per treatment is mentioned between brackets

[6] means with a different letter within a column and within each broiler line are significantly different from each other ($p<0.05$)

3.3. Carcase composition

Carcase lipid or protein content were not changed significantly by using feed restriction, although a trend of increasing lipid content was found in the second trial (Tables 44 and 45). In contrast, 308-birds in the first trial showed a near to significantly lower total lipid content after feed restriction. In all cases, the abdominal fat content was not changed significantly by restriction.

Pelleting feed had no significantly negative effect on carcase lipid or protein content, and even seemed to reduce the abdominal fat content (significant for Ross 508 in the second trial). Pelleting feed tended to have a positive effect on yield and especially breast meat percentage. Only for the 508-chickens in the first trial, the improvement was found to be significant for breast meat (Table 44).

Table 45 : Influence of feed restriction and structure on protein and lipid content (g/kg whole bird inclusive feathers), abdominal fat content, carcase yield and breast meat percentage (42 days of age) (mean ±SD) (Trial 2)

ROSS 308	**protein content**	**lipid content**	**abd. fat (g/kg)**	**yield (%)**	**breast meat (%)**
treatment					
ad libitum	180.4±2.5	143.4±4.3	23.1±3.4	68.1±0.9	28.2±1.3
restricted	180.3±3.5	150.6±9.0	22.9±2.2	67.7±0.7	27.5±1.6
feed					
mash	181.2±2.7	149.4±8.8	23.5±3.2	67.7±0.9	27.2±1.3
pellets	179.5±3.2	144.6±6.3	22.5±2.4	68.1±0.6	28.5±1.4
ANOVA (*p*-values)					
treatment	0.983	0.124	0.899	0.465	0.370
feed	0.419	0.279	0.568	0.409	0.154
ROSS 508					
treatment					
ad libitum	180.9±3.9	139.6±1.0	21.6±3.4	67.8±0.7	28.2±1.3
restricted	182.8±3.4	142.1±4.6	20.5±3.4	67.7±0.9	29.2±1.1
feed					
mash	182.1±3.3	138.3±4.2	23.4±1.8[a7]	67.3±0.4	28.1±1.2
pellets	181.7±4.3	143.5±9.9	18.7±2.7[b]	68.1±0.8	29.2±1.1
ANOVA (*p*-values)					
treatment	0.389	0.612	0.428	0.909	0.183
feed	0.864	0.302	0.010	0.083	0.853

[7] means with a different letter within a column and within each broiler line are significantly different from each other ($p<0.05$)

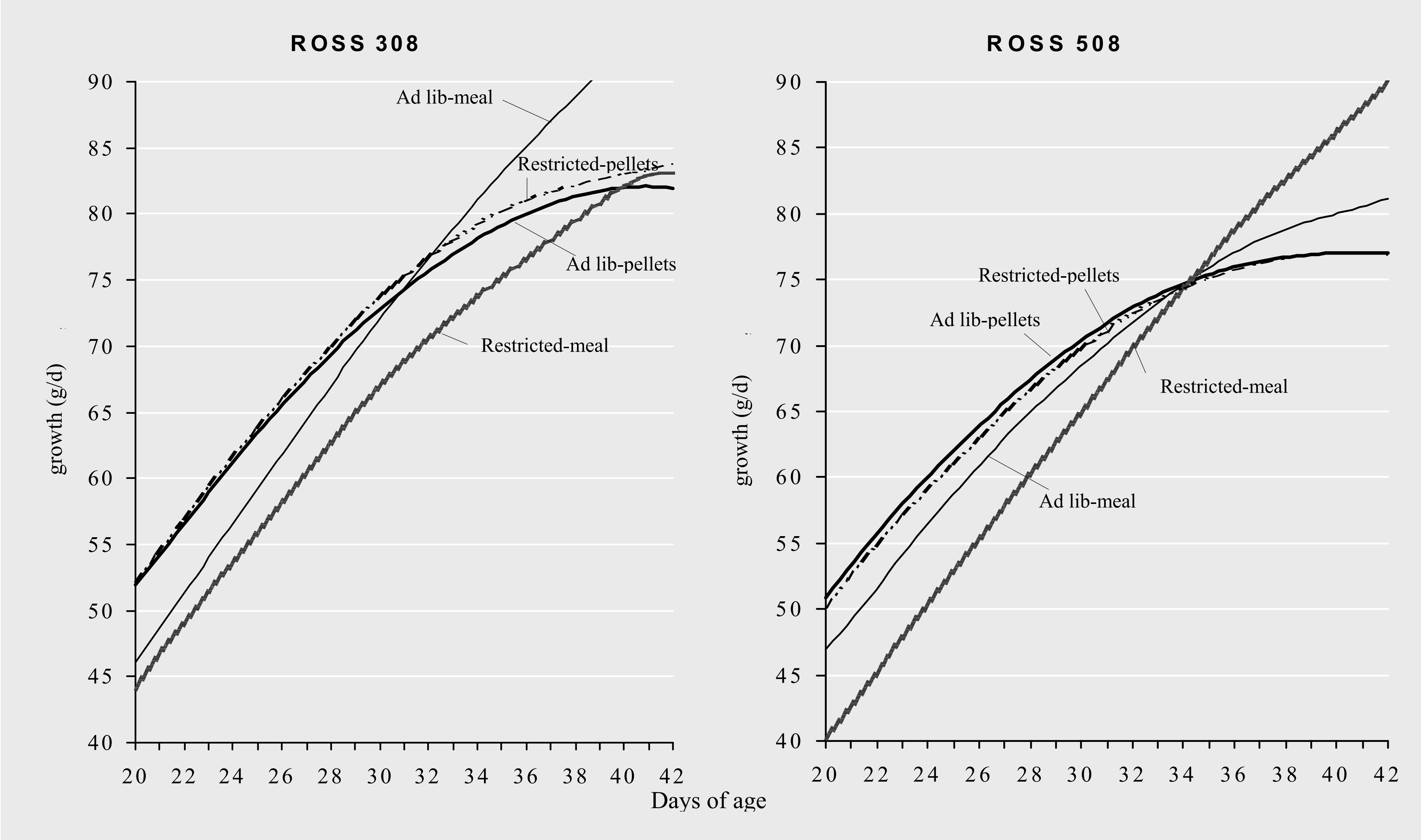

Figure 31 : Influence of feed restriction and feed structure on growth from 20 to 42 d of age (Trial 1)

Ross 308-chickens

Ad lib – meal : $W_t=45.9\ e^{(0.16303/0.03086)(1-e^{-0.03086t})}$ $R^2=0.997$

Restricted – meal : $W_t=45.9\ e^{(0.16483/0.032611)(1-e^{-0.032611t})}$ $R^2=0.994$

Ad lib – pellets : $W_t=45.9\ e^{(0.18273/0.03756)(1-e^{-0.03756t})}$ $R^2=0.999$

Restricted – pellets : $W_t=45.9\ e^{(0.1828/0.03742)(1-e^{-0.03742t})}$ $R^2=0.999$

Ross 508-chickens

Ad lib – meal : $W_t=42.2\ e^{(0.17792/0.03563)(1-e^{-0.03563t})}$ $R^2=0.998$

Restricted – meal : $W_t=42.2\ e^{(0.15741/0.02903)(1-e^{-0.02903t})}$ $R^2=0.993$

Ad lib – pellets : $W_t=42.2\ e^{(0.19043/0.03939)(1-e^{-0.03939t})}$ $R^2=0.997$

Restricted – pellets : $W_t=42.2\ e^{(0.18823/0.03876)(1-e^{-0.03876t})}$ $R^2=0.998$

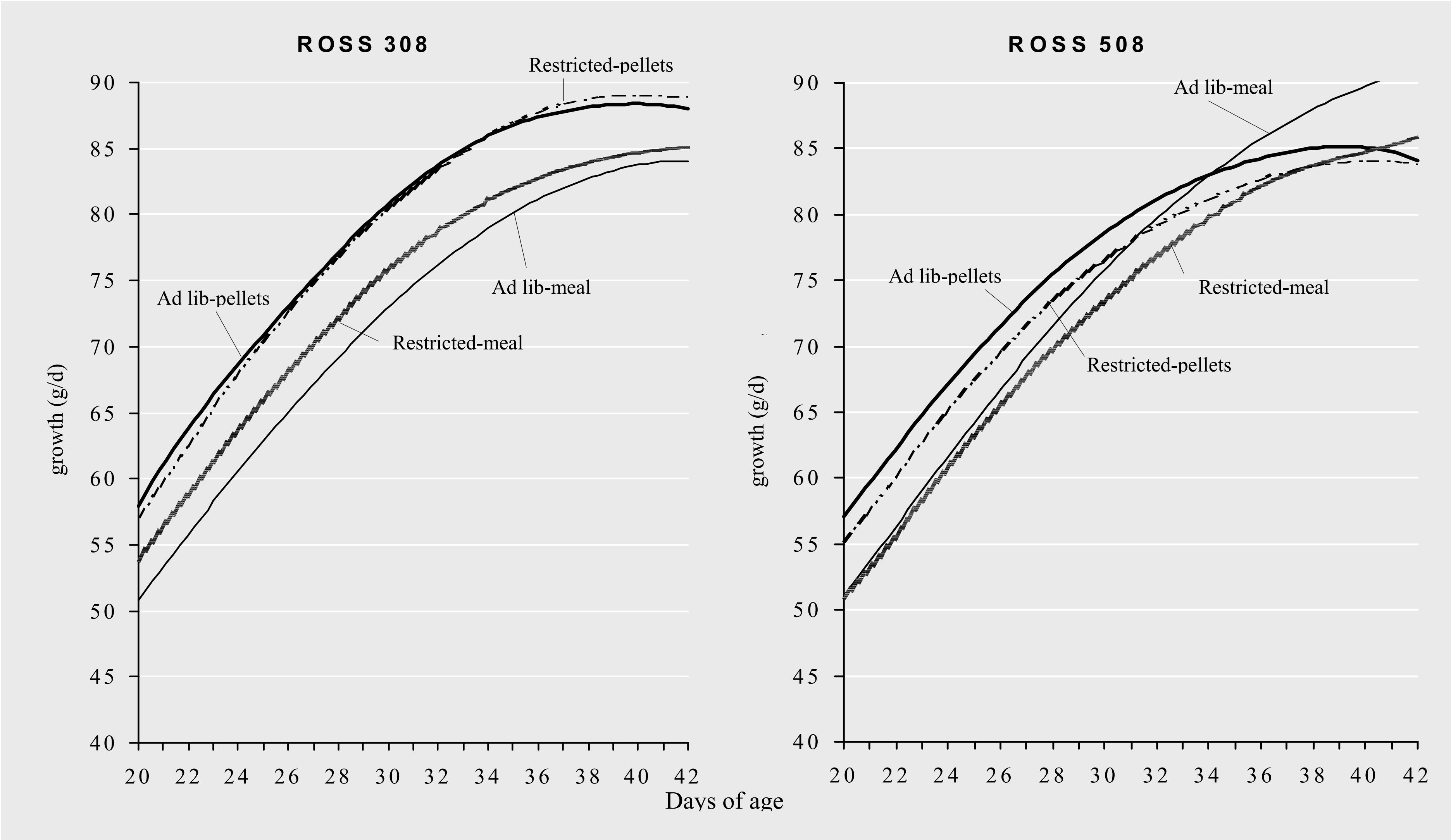

Figure 32 : Influence of feed restriction and feed structure on growth from 20 to 42 d of age (Trial 2)

Ross 308-chickens

Ad lib – meal : $W_t = 44.1\ e^{(0.18242/0.03687)(1-e^{-0.03687t})}$ $R^2=0.994$

Restricted – meal : $W_t = 44.1\ e^{(0.18809/0.03824)(1-e^{-0.03824t})}$ $R^2=0.993$

Ad lib – pellets : $W_t = 44.1\ e^{(0.19608/0.03986)(1-e^{-0.03986t})}$ $R^2=0.993$

Restricted – pellets : $W_t = 44.1\ e^{(0.19368/0.03917)(1-e^{-0.03917t})}$ $R^2=0.999$

Ross 508-chickens

Ad lib – meal : $W_t = 41.9\ e^{(0.18259/0.03569)(1-e^{-0.03569t})}$ $R^2=0.998$

Restricted – meal : $W_t = 41.9\ e^{(0.18454/0.03675)(1-e^{-0.03675t})}$ $R^2=0.998$

Ad lib – pellets : $W_t = 41.9\ e^{(0.19969/0.04069)(1-e^{-0.04069t})}$ $R^2=0.995$

Restricted – pellets : $W_t = 41.9\ e^{(0.19584/0.039813)(1-e^{-0.039813t})}$ $R^2=0.998$

In general, there seemed to be no significant negative effect of feed restriction on yield or the valuable part breast meat percentage when compensatory growth was complete. However, in

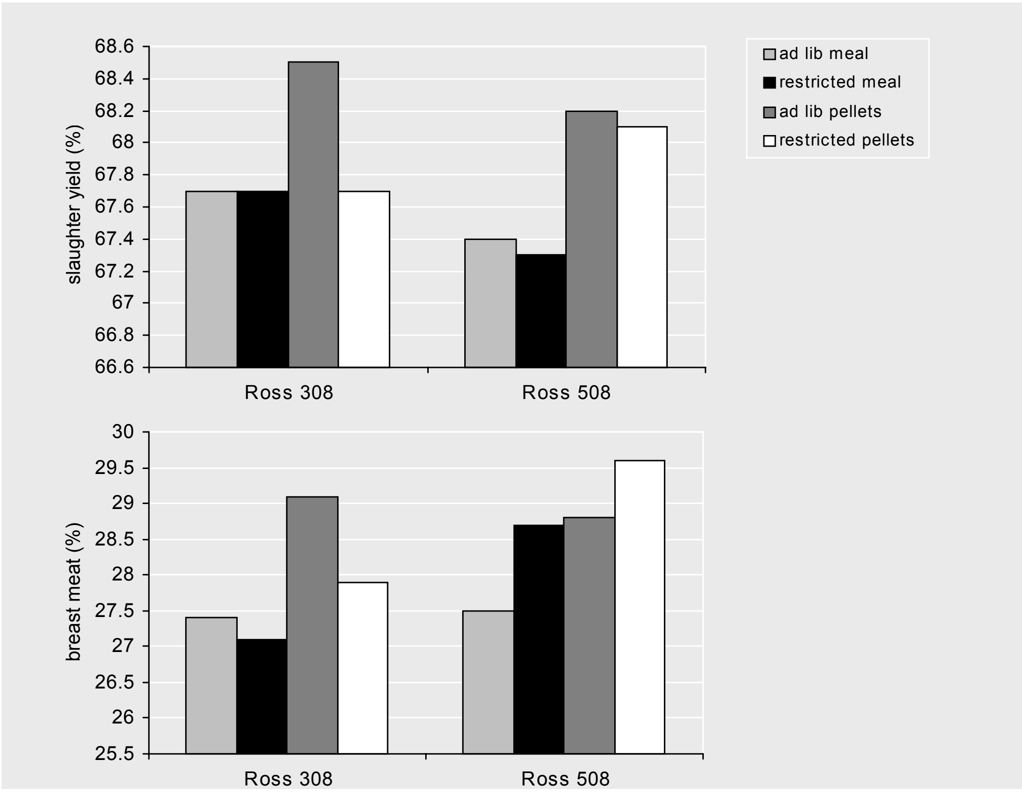

Figure 33 : Influence of feed restriction and feed structure on slaughter yield and breast meat percentage of Ross 308- and Ross 508 broilers (Trial 2)

the second trial, although compensatory growth was complete, there were some indications that when using feed restriction in combination with pellet feeding, losses in slaughter yield and breast meat percentage are possible (Figure 33). This was only the case for the 308-chickens. For the 508-chickens, the increased slaughter yield and breast meat percentage due to the use of pelleted feed was not lost by feed restriction (Figure 33).

4. Discussion

The general performances of the birds were lower in trial 1 in comparison with trial 2. Although protein and energy contents were constant in the diets of both trials, due to least cost formulation, some changes in amino acid ratios (relative to lysine) were found. Except for leucine, all ratios in the first trial (starter + grower) were close to the recommendations of the ideal amino acid profile (CVB, 1997; Lippens *et al.*, 1997; Mack *et al.*, 1999; Baker *et al.*, 2002). The somewhat higher ratio of leucine to lysine (general recommendation around 109) cannot be a reason for the somewhat lower results in trial 1. Indeed, in a study of Uzu (1993) even a ratio of 141 was recommended, which is much higher than in the current trial (125). Moreover, this same diet gave good results in the trial described in the previous chapter (Lippens and Huyghebaert, 2003). It is presumed that maybe environmental factors or a subclinical infection had an influence on the general final performances of the birds.

When restricting birds to 80 % of the *ad libitum* intake, starting from day 4 for 4 days, the growth curve can be made more concave. Body weights shortly after the restriction (day 8) were 12 to 16 % (approximately 20 g) lower in comparison with the concomitant *ad libitum* group. However, this was not sufficient to induce compensatory growth in all cases.

The variability in compensatory growth capacity found is not new. It has been suggested that, in addition to the nature, timing, severity or duration of the restriction, strain, line and sex and also age of the parent stock play a role in compensatory growth capacity (Lippens *et al.*, 2000; Lippens and Huyghebaert, 2003). Sex ratios were close to a 50:50 distribution (males/females : 51/49, 52/48, 55/45 and 51/49 for Ross 308-trial 1, Ross 508-trial 1, Ross 308-trial 2, Ross 508-trial 2). Moreover, ratios within each trial and line were not significantly different.

On the other hand, differences between both lines were very pronounced within each trial. This was confirmed by the many interactions in the current trials (not shown) and earlier observations (Lippens, 2001; Lippens *et al.*, 2002a, 2002b, 2002c; Lippens and Huyghebaert, 2003). As both lines are genetically very different, these interactions were expected. However, differences were not consistent between both trials. There are no indications that differences in diet formulation had an influence on this variability. As mentioned before, there was no clear reason for the differences in performances between trials.

An important factor of variance in describing compensatory growth capacity seems to be feed structure, as demonstrated in the current trial. The restrictions used, when using pelletised feeds, were insufficient to induce compensatory growth. As the relative reductions shortly after the restriction were identical to mash feeding, it indicates that birds fed pellets recovered more easily. They realised the same growth after the restriction period as the controls. With this equal growth, they finished at a final body weight not significantly different from the controls. With meal fed birds, however, compensatory growth was found in some cases. It is not clear why compensatory growth was that variable although circumstances are similar.

Using pellets instead of mash, zootechnical performances and carcase characteristics were improved. Growth of birds fed pelleted feeds was 6 to 7 % higher in comparison with mash feeding. These findings are in agreement with results of Plavnik *et al.* (1997), Hamilton and

Proudfoot (1995) and Urdaneta-Rincon and Leeson (2002). These higher growth rates were partially linked to increased feed consumption. However, in some cases, feed was also converted more efficiently to growth.

High juvenile growth rates, common for the modern broiler lines and stimulated by pellet feeding, are to be expected to inhibit an optimal development of the vital organs. This induces an increase of the occurrence of metabolic diseases. According to Nir *et al.* (1995), increased mortality observed in birds fed pelleted diets might also be linked to a reduced bird activity, because pellet feeding makes the bird spend less time and energy on feeding. In the current trial no significant increase in mortality was found, which is in agreement with the results from Jones *et al.* (1995). However, variability was higher.

Using restriction programmes, in accordance with the previous results, a general tendency of a reduced mortality was found and again, no significant differences could be established. As mentioned before, the relatively low number of birds in the trials might explain this. Moreover, in both trials, there was only a very low number of chickens which died from SDS and ascites (trial 1: 3 and 3, respectively; trial 2: 8 and 2, respectively). This makes it very hard to evaluate influences of feed structure or restriction (or possible interactions) on metabolic diseases.

During this period of early restriction it is stated in the literature that the number and size of adipocytes is reduced (Meluzzi *et al.*, 1998). This makes it possible to lower carcase and abdominal fat content in the finishing bird. However results on fat deposition are very inconsistent. In some publications, the lowered fat deposition could be confirmed (Plavnik and Hurwitz, 1985, 1988, 1991; Jones and Farell, 1992b). Others reported reductions with concomitant body weight losses or no reduction at all (Mollison *et al.*, 1984; Cabel and Waldroup, 1990; Mudrić *et al.*, 1994; Pinchasov and Jensen, 1989; Leeson *et al.*, 1991; Fontana *et al.*, 1993; Scheideler and Baughman, 1993; Susbilla *et al.*, 1994; Deaton, 1995; Palo *et al.*, 1995; Cristofori *et al.*, 1997). No significant differences in body weight or fat deposition (total fat and abdominal fat content) were found in the current trials.

According to literature however, pelleting feed seems to induce an increase of the abdominal fat pad and total fat content (Nir *et al.*, 1994; Plavnik *et al.*, 1997). Indeed, using pellets, the energy allotted to maintenance seems to be reduced because of a combined effect of a higher intake in the form of pellets (lower bulk density) on the one hand and a reduced activity of the birds on the other hand. This would mean a higher metabolisable-into-net energy conversion, thereby inducing an increased body fat deposition (Nir, 1997). In the current trial, only a tendency for a higher total lipid content was found. On the other hand, abdominal fat, in some cases, tended to be reduced. In general, it is stated that different fat depots in the body are positively correlated (Leenstra, 1986). These conflicting results are probably due to a high variability in between animals combined with a relative low number of replicates.

In agreement with earlier findings, it was observed that with incomplete compensatory growth, losses in slaughter and breast meat yield are possible (Lippens *et al.*, 2000; Lippens *et al.*, 2002a). Results, however, could not confirm the hypothesis that feed structure has a major effect on slaughter yield and breast meat percentage of feed restricted birds. There was a trend, only for the Ross 308-birds in trial 2, of losses in slaughter yield and breast meat percentage, even though final body weights were not impaired, when combining feed restriction with pellet feeding. More research is needed to verify these findings.

It can be concluded that many factors can play a role in the ability of the bird to realise compensatory growth after early, temporary feed restrictions. One of the factors playing a role is feed structure. A restriction to 80 % of the *ad libitum* intake of the previous 24 hours, starting from day 4 for 4 days, can give good results when using mash diets. Final body weights do not deviate significantly from the controls. Economical advances can be realised mainly by lowered mortality. No negative effect on carcase yield or breast meat has to be expected, when compensatory growth is complete. However, when combining the same feed restriction with pellet feeding, no compensatory growth is realised. It seems that in these circumstances a more severe restriction is needed. It is not clear if these more pronounced restrictions with pellet feeding will induce sufficient compensatory growth to prevent losses of slaughter yield or breast meat percentage. More research is needed to confirm this.

General conclusions and future perspectives

At first sight, when looking at the results of feed control on the growth pattern of broiler chickens, conclusions may be considered to be rather disappointing. Indeed, it is clear that a lot of variation is common. Growth retardation short after the restriction (8 d of age) ranged from 7 to 15 % of the control group when using a restriction to 80 % of the *ad libitum*-intake for 4 days starting from day 4. Differences in final body weights between restricted and *ad libitum* fed birds, ranged from –5 to +3.7 %. No correlation was, however, found between 8-d old weight after retardation and catch-up capacity.

Using feed control programmes has, indeed, the potential of increasing the performances of broiler chickens (up to 3.7 % higher final body weight, up to 1.5 % better FC). Moreover, mortality and leg disorders can be reduced. However, due to the rather small numbers of broilers used, few significant changes in mortality and especially in the occurrence of metabolic diseases could be found. Still, total mortality (death+removed) in the current work was on average lowered with 2.5 % due to the feed restrictions. Although significance is lacking, in practice, these percentages have an important economic value. It can be concluded that the described restriction programmes might be an economically feasible management tool for the nowadays available modern broiler lines and the current applied management techniques although results are rather variable.

When compensatory growth is pronounced and catch-up growth more than compensating, an increased breast meat percentage seems possible. On the other hand, the effect of feed restrictions on fat deposition in the carcase is rather variable. Mostly however, changes in carcase or abdominal fat content were not significant in the present work. It can be concluded that no significant negative effects on carcase- and meat quality should be expected with feed restricted broilers, especially when compensatory growth is complete. On the other hand, the hypothesis stating that growth control can reduce fat deposition could not be confirmed in this work.

Next to the reduced occurence of metabolic disorders, with the associated pain and suffering, and the reduced mortality, the restrictive feeding programmes can also increase N-retention (1-2 %). In this way, growth control also contributes to a solution for the manure problem and environmental pollution.

Very remarkable are the findings that the catch-up capacity of the Ross 508-chickens is sometimes very pronounced (up to 3.7 % higher final body weights). Although this line is already characterised by a retarded juvenile growth rate, still it seems susceptible for an additional growth control. Using Ross 508 broilers in future opens the possibility to further optimise the production process by, primarily management and secondly also by genetics.

A lot of factors of impact involved in compensatory growth capacity are considered in the present work. Still it seems, in the given situation, not possible to predict the final results of a given feed restriction programme. In other words, for the farmer, it is very difficult to decide whether or not to choose for a feed restriction programme at the arrival of the chickens. No guarantee of economic returns can be given in advance, because it is not clear in what circumstances complete catch-up growth can be realised.

A possible solution for this problem might be found in a continuous monitoring of the growth trajectory of broiler chickens; including deviations between actual growth and the desired trajectory. The regulation of feed intake seems to be a useful management tool for adjusting the growth curve. In the framework of the current work, a model-based growth algorithm has been developed relating continuously the growth response to the control input 'feed intake' with a feedback possibility up to 5 days (Aerts *et al.*, 2003a,b).

When controlling the growth of the birds, a target trajectory should be selected. This is not easy and depends on the environmental conditions. Based on the findings in the current work, an advice for Ross 308 and Ross 508 broilers is given in Table 46 and Figures 34-35. This is only applicable for broilers of mixed sexes fed with meal diets, in a standard temperature and lighting schedule (see chapter 3). Based on the personal experience of the farmer, this model could be further optimised in relation to the typical environmental conditions of his broiler farm.

Of course, there are some limitations in the growth control of birds. Birds can only grow within their biological capacity. Several factors, as one-day-old chick quality, health status or environmental conditions can impair their capacity to follow the target growth curve. Naturally, management must be optimised to avoid these problems as much as possible.

When the developed model-based control algorithm is integrated in the computer software, available on the farm, no major efforts will be necessary from the farmer to further optimise his production process. In the future, it would even be possible to link this information available on the farm to the information network of the slaughterhouses. In this way, slaughterhouses would have an overview of how many birds with that particular mean weight are available at a certain date. With this information, also slaughterhouses could optimise their activities. Maybe the farmer could get some monetary compensation for this additional information.

Table 46 : Advices for a target growth trajectory for Ross 308 and Ross 508 chickens using on line-growth control[1] on meal diets

Days of age	Ross 308	Ross 508
4	87	80
5	94	87
6	106	99
7	119	112
8	135	128
9	153	145
10	172	164
11	193	185
12	216	208
13	241	233
14	270	260
15	303	290
16	339	323
17	378	359
18	420	399
19	464	442
20	511	488
21	562	538
22	617	591
23	675	647
24	737	707
25	802	770
26	870	835
27	940	902
28	1012	971
29	1086	1042
30	1162	1115
31	1241	1190
32	1323	1268
33	1407	1349
34	1493	1433
35	1581	1519
36	1670	1607
37	1760	1697
38	1851	1789
39	1943	1882
40	2036	1977
41	2130	2073
42	2225	2169

In Belgium no extra compensation is provided for increased breast meat percentages. However, with the model-based control of the growth, in a further stage of development, some guarantees could be given concerning breast meat percentage of the delivered birds. If the farmers were paid for this information, the economic returns would further increase.

Moreover, more research is necessary to implement other factors as feed quality or least-cost rations in such a model to further optimise the process in an economic way (Aerts *et al.*, 2003b). One of the possibilities for future development may be the optimisation of protein (AA) intake. In recent studies on amino acid requirements of broilers, it has become clear that protein/AA-content of the diet should be adjusted in function of age (Fisher, 2002; Geraert *et al.*, 2002). Indeed, until now, AA-requirements are determined for rather long periods of time (2 to 3 weeks of age). However, the recent research stresses the need for more accurate requirements in function of age (daily) to optimise performance and N-retention. Geraert *et al.* (2002) suggested using the actual performance of the bird to determine daily lysine-requirements.

[1] on line-growth control starts at day 4

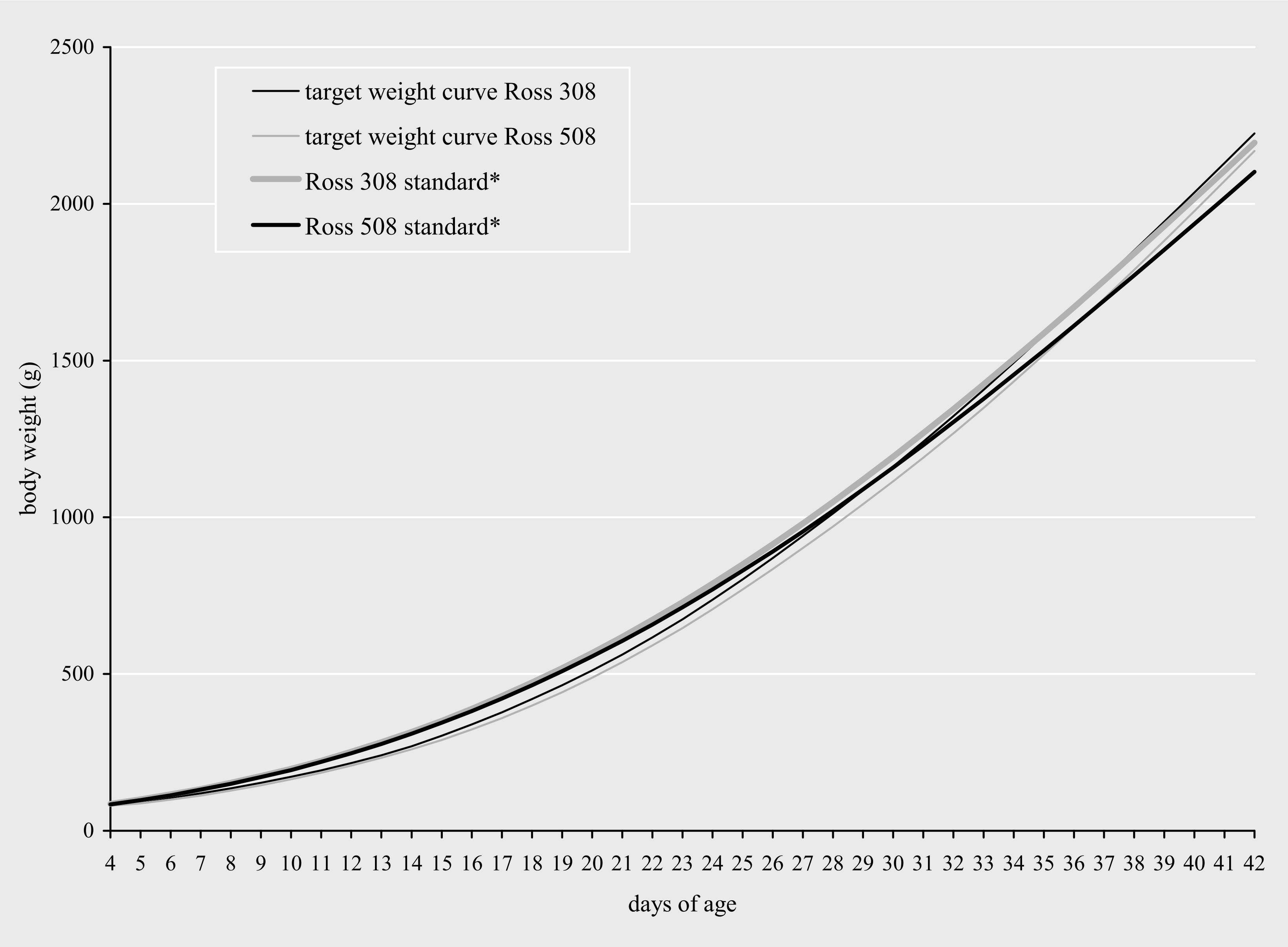

Figure 34 : Target and standard weight trajectories of Ross 308 and Ross 508 chickens in function of age

* standard weight trajectory in current trial circumstances

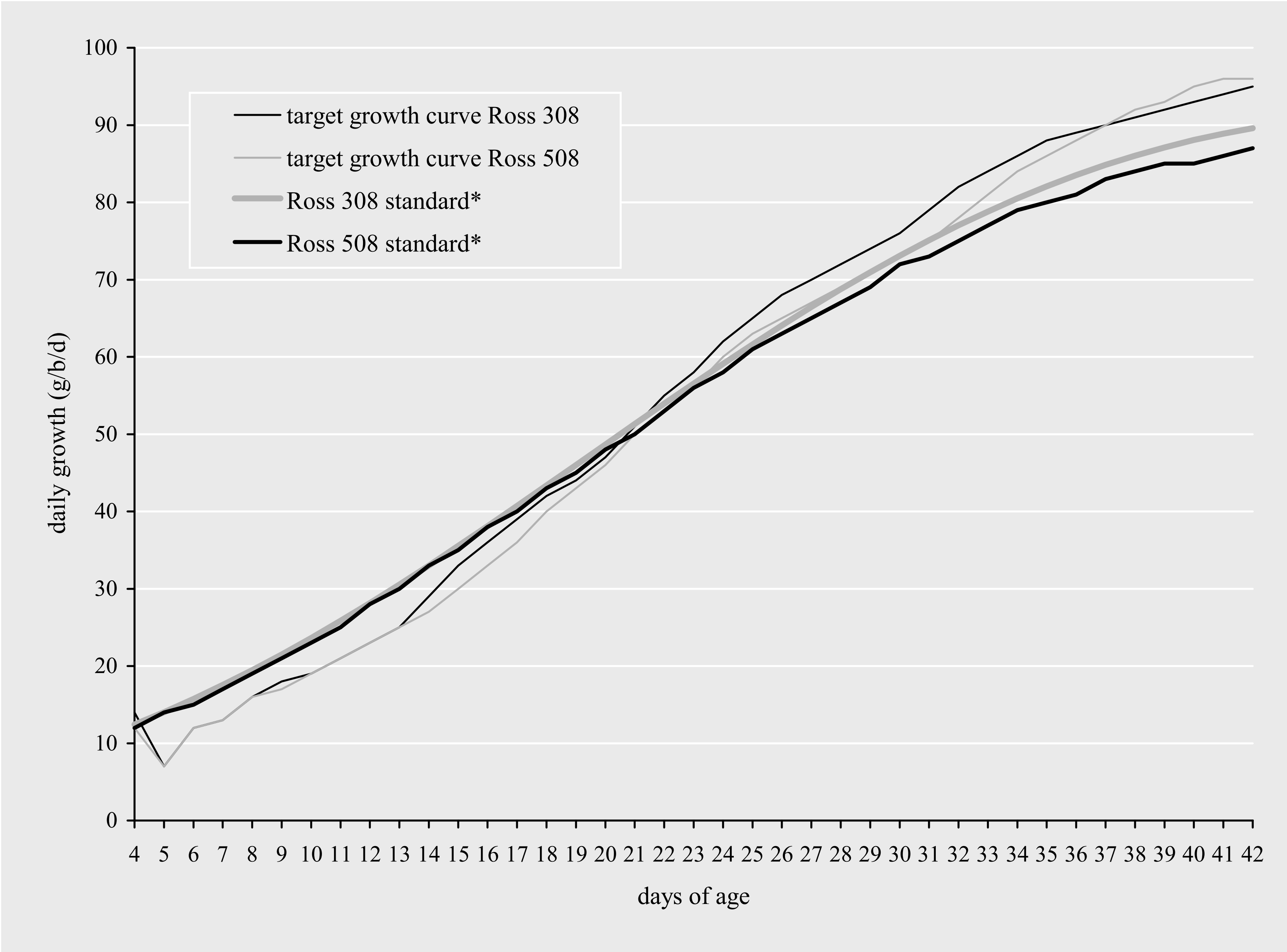

Figure 35 : Target and standard growth curves of Ross 308 and Ross 508 chickens in function of age

* standard weight trajectory in current trial circumstances

According to these authors, the requirement for e.g. lysine can be calculated according to the following equation (based on a literature overview) :

$$Y = 0.01911\,x - 0.1179$$

with Y = available lysine intake (g/d)
x = body weight gain (g/d)

In this way, not only quantity but also the quality of the feed could be optimised daily. In practice, this could be realised by mixing two different diets (e.g. high protein meal and whole grains). Based on model calculations, the quantity and the desired levels of the available diets can be calculated and distributed through the automatic feeding system to feed the birds. However, in every stage, the advantages of introducing more factors to the model should be evaluated against the increased complexity.
The model-based technique could also be extended to other animal species. As the same problems with metabolic diseases are existing in e.g. the turkey industry, it would be interesting to extend the model to control the growth of turkeys also.

Summary

Genetic selection in meat-type chickens has provided the industry with flocks which reach the target slaughter weight in a shorter period of time. Indeed, modern broiler strains are characterised by a very high growth rate and a low feed conversion ratio. However, some unfavourable selection responses have also occurred. These modern meat-type broilers show an increased fat deposition, a higher incidence of leg problems and a greater susceptibility to metabolic diseases such as 'sudden death syndrome' (SDS) and ascites. These negative aspects of selection are of major concern for the farmer and processor, because they cause important economic losses.

To deal with these problems, early-life feed restriction programmes were designed. Using these programmes, broilers are restricted in growth in the early phase of life. In this way, they get the opportunity to develop their vital organs and skeleton in a more optimal way. Relying on the phenomenon of compensatory growth, equal final body weights can be achieved. In addition, better feed utilisation and lower carcase fat contents are expected. In literature, it was confirmed by a number of researchers that losses due to metabolic diseases could be limited in this way.

To limit growth, feed intake can be restricted in a quantitative or a qualitative way. When a quantitative restriction is used, the quantity of feed is restricted to a certain level during a short period of time early in life. When using a qualitative restriction, birds are able to keep on eating *ad libitum* but get, during similar periods, a feed of a minor quality.

A number of trials describing such feed restriction programmes are available in literature. However, literature is not conclusive on the effects of these programmes on zootechnical performances and carcase composition. Moreover, as the progress in broiler genetics is very intensive, new information on the effect of early feed restriction on the currently used strains or lines is very important for the poultry industry. New information is provided by the results of the current work. Moreover, results on the effect of feed restriction on meat quality are almost non-existing in literature. Research on the effect of feed restriction on the chicken meat quality is incorporated in the current work.

In chapter 4 and 5 quantitative and qualitative feed restriction techniques are evaluated on a Ross 508 and a Hybro G strain. In the first trial (chapter 4), birds were restricted to 80 % or 90 % of *ad libitum* intake (of the previous 24 h of the control group) for 4 d or to 80 % for 8 d. All restrictions started on d 4. In chapter 5 two qualitative feed restrictions were tested. A low energy and a NaCl-deficient diet were used.

Results indicated that a restriction programme should not last longer than 4 days to give the birds the opportunity to recover completely. However, when using a quantitative restriction,

only the 90 % restriction resulted in a complete catch-up at 42 days of age. The qualitative feed restrictions used, seemed to be a good way to induce a compensatory growth after the temporary growth retardation. Indeed, final body weights of the chickens subjected to these restrictions showed no significant differences with the control birds. However, in contrast with some findings in literature, no significant reduction in feed conversion or fat content was found.

Probably due to the rather low number of animals used in these kind of trials, no significant reduction of losses due to metabolic diseases were recorded. Only a positive trend on mortality and losses due to SDS was found. Although differences were not statistically significant, still the economical impact of these figures can be important when working with a large number of birds in field circumstances. No conclusions can be drawn for the factor ascites as a very small number of birds died of this metabolic disease.

There was no indication in these trials that changes in growth trajectory (growth retardation - compensatory growth) induced by quantitative or qualitative feed restriction had a negative effect on slaughter yield or cut-up parts when compensatory growth was complete. Meat quality was described in terms of pH_{24} (24 h after slaughter), meat colour, moisture, drip-%, cooking losses and shear force. Considering these factors, there were no indications of deteriorating meat quality with feed restricted broilers.

It was concluded that a quantitative feed restriction to 90 % of the *ad libitum* intake or a qualitative feed restriction (as used in the current work), give some indications of being practical tools to reduce losses due to metabolic diseases without deteriorating performance, carcase or meat quality. However, some additional research at our Institute revealed that, in many cases, the restriction to 80-% (4 d) also has the ability to induce sufficient compensatory growth.

In a second part of this work, the nitrogen retention during compensatory growth was examined. Indeed, with compensatory growth, an improved efficiency is expected. As protein is one of the most expensive components in the cost of a complete diet, it is important to use protein in particular as efficient as possible. Moreover, a more efficient use of N contributes to the alleviation of environmental pollution.

In chapter 6, two experiments were conducted to investigate the influence of compensatory growth on the efficiency of nitrogen retention in two lines of Ross broilers, Ross 208 (308) and Ross 508. The Ross 208 or 308 (replacing the 208-line completely since September 2000) is the commonly used commercial line of Ross. The Ross 508-line used in most of the following trials however, is already genetically selected for a lower initial growth rate in view of reducing metabolic diseases. Ross 508 broilers also are characterised with a significant higher breast meat percentage. As it is interesting to know in what way this modified line can still have an advantage of restricting growth additionally by feed restrictions, intensive research on this line is presented in this work.

Based on the results of the previous trials and some additional unpublished work conducted at our Institute, it was decided to use an restriction to 80 % for 4 days starting from day 4 in all subsequent trials. After the period of restrictions all birds were fed *ad libitum*.

In the trials of chapter 6 it was indeed confirmed that this kind of quantitative feed restriction is a good tool to induce sufficient compensatory growth. In both trials, the final body weight of the restricted Ross 208(308) birds was similar or even higher than the weight of the control group. For the Ross 508 line, compensatory growth was substantial in the first trial, but non-existing in the second trial. In contrast with the results of the previous chapters, feed conversion could be improved significantly by restriction. However, in confirmation with the results of chapter 4 and 5, only a tendency for lowered mortality was found.

As compensatory growth was established, some improvement in N retention was induced. Although differences were not significant, they can be environmentally important. Moreover, N retention seemed to be correlated with broiler line and age of the birds.

Little references in literature are known concerning the protein requirement before and during the period of compensatory growth. In chapter 7 research was done on the effect of protein content of the starter diet on the one hand and the protein content of the grower (during compensatory growth) on the other hand. In literature it became clear that lowering protein content of the starter had no detrimental effects on subsequent broiler performances. It was, however, not clear if lowered protein content of the starters impairs the ability of restricted birds to compensate for growth retardation. A standard starter diet with 22 % CP was compared with a low protein starter of 20 % CP in chapter 7.

To investigate the need for higher protein contents during compensatory growth, a normal grower diet (20 % CP) was compared with a high protein grower of 22 %, within both starter diets. Again the two lines of the Ross strain were set up in the trial and the same quantitative restriction was established.

Lowering the protein content of the starter diet did not change the growth performance or compensatory growth capacity of the broilers significantly although a tendency for a worsened FC was found. The fact that a quantitative feed restriction prevented this deteriorating FC, can probably be explained by the earlier findings that a better N-utilisation and a reduced protein turn-over can be expected when birds are retarded in their initial growth and compensatory growth is induced. A higher protein content of the grower could further ameliorate FC significantly. Especially Ross 508 birds fed a low protein starter seemed to have a higher need for protein during the 'catch-up'-phase.

The influence of dietary treatment on carcase composition is quite different between the two lines. For the Ross 308 birds, carcase quality was clearly more affected by early feed restriction than by grower protein content. The early feed restriction had a significantly positive (lowering) effect on total fat content (g/kg whole bird) but only a limited positive effect on abdominal fat content and breast meat percentage. Again, Ross 508 chickens seemed to have their benefit of receiving a high protein grower after early feed restriction. Indeed, the dietary grower protein content had positive effects on total carcase protein content and carcase yield.

The above mentioned results confirm the findings in literature that the effect of an early feed restriction can be rather variable. This can partly be explained by the fact that a lot of factors are of influence when considering this kind of growth control. Nature, timing, severity and duration of the restriction or genetic factors such as strain and sex are already described in literature and further illustrated in the current work. However, eliminating these factors, still results of feed restriction programmes are rather unpredictable. In other words, these parameters are not sufficient to explain all of the variation found. When looking for further explanations for the sometimes variable results, some additional factors of possible influence are described in the chapters 8 and 9.

The results of the described trials above gave some indications that "chick quality" may have an influence on the capacity of the chickens to establish compensatory growth. In chapter 8, research was done looking at the influence of breeder age, as a possible indication for difference in chicken quality, on the compensatory growth capacity of their progeny broiler chickens.

Indeed, in literature, it was found that a correlation exists between one-day old broiler weight and growth capacity. No research however has been done on the influence of day-old broiler weight on compensatory growth capacity of feed restricted birds. In other words, the question arises if measurements of one-day old chicken weights are related with their 'catch-up' growth capacity when birds are feed restricted at an early age.

Three different ages of parent stock were used in two subsequent trials with Ross 308 and 508 birds. Chickens from young (± 32 weeks), middle age (± 45 weeks) and old (± 60 weeks) parent stocks were used in both trials. These differences in breeder age induced some

significant differences in one-day old chicken characteristics. So, it was assumed that in this way also chicken quality was differentiated.
The influence of breeder age on compensatory growth capacity was variable between the two subsequent trials. Chickens of the young parent stock showed a pronounced compensatory growth in the first trial, which was not confirmed, in the second trial. Furthermore, there were indications that a more severe restriction might be needed for the chickens from the older parent stock. It is clear from these results that age of parent stock influences compensatory growth capacity, but the differences are still depending on trial circumstances.
In chapter 9 the influence of feed structure, as another factor of variance, on growth capacity was examined. Two experiments with both Ross lines were conducted to evaluate the possible difference in compensatory growth capacity after a quantitative feed restriction using either mash or pellets. The ages of the parent stock were in the first trial 46 and 44 weeks for Ross 308 and Ross 508, respectively. For trial 2, both parent stocks aged 43 weeks of age.
In both trials, and for both lines, restriction did not influence final body weight and FC significantly. On the other hand, pelleting feeds gave higher final body weights and better feed conversions in comparison with mash feeding. Compensatory growth was lacking when using pellet feeds. In contrast, when using mash feeding, compensatory growth was found, although not in all cases. Also the age at which compensatory growth started was variable.
Notwithstanding the increased growth rate due to pellet feeding, no increased mortality was found. Feed restrictions, however, induced again a trend of lowered mortality. There were no indications of a correlation of this positive effect with the feed structure.
No consistent effects of feed restrictions on carcase composition were found. On the other hand, pellets had a positive effect on yield and breast meat percentage. However, in the second trial, there were some indications with the 308-line only, that when using feed restrictions in combination with pellet feeding, a loss in slaughter yield and breast meat percentage is possible.
It was concluded that many factors can play a variable role in the ability of the bird to realise compensatory growth after early, temporary feed restriction. Moreover, it seems impossible to ameliorate FC and carcase fat content in all circumstances. The variation found can be partially explained by feed structure or “chick quality”. Still it seems, in a given situation, impossible to predict the final results of a given feed restriction programme. In other words, for the farmer, it is very difficult to decide whether or not to choose for a feed restriction programme with the arrival of the chickens. It might be advised to control growth

continuously in time by adjusting feed intake from day to day rather than using an a priori determined feed restriction in all circumstances.

Samenvatting

(Dutch summary)

De genetische selectie in de vleeskuikensector heeft ervoor gezorgd dat vleeskuikens op steeds vroegere leeftijd slachtrijp zijn. Aan deze hoge groeisnelheid en daarmee gepaard gaande verbeterde voederconversie zijn echter een aantal negatieve responsen gekoppeld. De moderne vleeskippenrassen worden gekenmerkt door een verhoogde vetaanzet (bij gelijke leeftijd) en het frequenter voorkomen van pootgebreken en metabole aandoeningen zoals ‘sudden death syndrome’ (SDS) en ascites. Deze negatieve selectieresponsen zijn zeer belangrijk gezien zij, zowel voor de kweker als bij de verdere verwerking, aanleiding kunnen geven tot uitgesproken verliezen.
Een manier om deze nadelige effecten te vermijden is het gebruik van een voederrestrictie. Bij het toepassen van dergelijke voedersturing wordt de jeugdgroei afgeremd waardoor de dieren de kans krijgen om hun vitale organen en skelet op een meer optimale manier te laten ontwikkelen. De daaropvolgende inhaalgroei (of compensatorische groei) zou voldoende moeten zijn om het beoogde eindgewicht te bereiken. Er wordt bovendien verondersteld dat deze verschuiving van de groeicurve aanleiding geeft tot een verbetering van de voederconversie en een verlaging van de vetaanzet. In de literatuur werd het positief effect van deze voederrestricties op uitval door metabole aandoeningen reeds aangetoond.
Om de groei te remmen kan gebruik gemaakt worden van een kwantitatieve of kwalitatieve voederbeperking. Wanneer een kwantitatieve voederbeperking wordt toegepast, wordt de dieren dagelijks een afgewogen hoeveelheid voeder gegeven gedurende een korte periode in de jeugdfase. Bij een kwalitatieve voederbeperking kunnen de dieren *ad libitum* blijven eten maar wordt een voeder met lagere kwaliteit voorzien gedurende de restrictieperiode.
Heel wat resultaten betreffende voederbeperkingen bij vleeskippen zijn reeds beschikbaar in de literatuur. De beschreven effecten op de zoötechnische prestaties en karkassamenstelling zijn echter nogal uiteenlopend. Bovendien moet er rekening gehouden worden met de zeer snelle evolutie in de genetica van vleeskippen. Nieuwe informatie betreffende de effecten van voedersturing bij de huidige vleeskippenrassen zijn daarom zeer belangrijk voor de pluimveesector. Nieuwe informatie wordt verschaft in het huidige werk. Bovendien blijkt uit literatuur dat er nog weinig geweten is over de vleeskwaliteit van voederbeperkte vleeskippen. Uitgebreid onderzoek werd daarom gedaan in het kader van dit doctoraatswerk.
In hoofdstuk 4 en 5 werden kwantitatieve en kwalitatieve restricties getest bij een Ross 508-lijn en een Hybro G lijn. In de eerste proef, beschreven in hoofdstuk 4, werden dieren beperkt tot 80 % of 90 % van de *ad libitum* opname (van de voorbije 24 u van de controlegroep) voor 4 dagen of tot 80 % voor 8 dagen. Alle beperkingen startten op dag 4. In hoofdstuk 5 werden

twee kwalitatieve beperkingen uitgetest op basis van zowel een lage energie-inhoud als een laag NaCl voeder.

Uit de resultaten bleek dat een voederrestrictie niet langer dan 4 dagen mag duren opdat de dieren de mogelijkheid zouden krijgen om voldoende inhaalgroei te kunnen realiseren. Enkel de beperking tot 90 % gedurende 4 dagen kon echter aanleiding geven tot een volledige ‘catch-up’ op 42 dagen leeftijd. De kwalitatieve beperkingen daarentegen bleken in beide gevallen goede mogelijkheden om voldoende compensatorische groei te induceren na de beperking. De gemiddelde gewichten van de beperkte dieren waren niet significant verschillend van de controlegroep. In tegenstelling met sommige resultaten in de literatuur kon de VC en het vetgehalte in geen enkel geval significant worden verlaagd.

Er kon geen significante daling van de sterfte worden aangeduid. Dit heeft waarschijnlijk te maken met het relatief laag aantal dieren dat in deze proeven werd gebruikt. Er kon enkel een trend van verminderde sterfte en uitval als gevolg van SDS worden vastgesteld. Hoewel de afnamen statistisch niet significant waren, kunnen zij toch een belangrijke economische impact hebben in praktijkomstandigheden met grote groepen dieren.

Er waren in de besproken proeven geen aanduidingen dat de gewijzigde groeicurve (groeivertraging – compensatorische groei) door zowel kwantitatieve als kwalitatieve voederbeperkingen aanleiding heeft gegeven tot negatieve effecten op vlak van slachtrendement of versnijdingsresultaten in de gevallen waar de compensatorische groei voldoende was. De vleeskwaliteit werd gemeten door bepaling van de pH_{24} (24 u na het slachten), de vleeskleur, vochtverlies, drip-%, kookverliezen en scheurkracht van het vlees. Metingen van deze factoren gaven geen aanduidingen van een verminderde vleeskwaliteit bij voederbeperkte dieren.

Er kan worden geconcludeerd dat een kwantitatieve voederbeperking tot 90 % van *de ad libitum* opname of een kwalitatieve voederbeperking, zoals toegepast in het huidige onderzoek, een praktisch toepasbaar middel blijken om verliezen als gevolg van metabole ziekten te verminderen zonder in te boeten op zoötechnisch prestaties of karkas- en vleeskwaliteit. Er moet echter vermeld worden dat bijkomende onderzoeksresultaten op ons Instituut aantoonden dat ook en vooral een beperking tot 80 % goede mogelijkheden biedt om de dieren kwantitatief te gaan beperken.

In een tweede deel van dit werk werd de stikstofretentie gedurende compensatorische groei bekeken. Tijdens compensatorische groei wordt inderdaad een verbeterde efficiëntie verwacht. Gezien eiwit een van de duurste componenten van het voeder is, is het zeer belangrijk om met name eiwit zo efficiënt mogelijk te gaan benutten. Bovendien kan op die manier bijgedragen worden tot een verminderde N uitstoot in het milieu.

In hoofdstuk 6 werden twee experimenten uitgevoerd om de invloed van compensatorische groei op de N-efficiëntie bij twee Ross lijnen, Ross 208 (308) en Ross 508, na te gaan. De Ross 308 (als opvolger van Ross 208 sinds september 2000) is de algemeen gebruikte, commerciële lijn van Ross. De 508-lijn daarentegen is genetisch reeds geselecteerd op een vertraagde jeugdgroei met het oog op de vermindering van metabole aandoeningen. Bovendien wordt de 508-lijn gekenmerkt door een verhoogd borstvleespercentage. Het was echter interessant om te onderzoeken of deze gewijzigde lijn nog een additioneel voordeel kan hebben van voederbeperkingen. Intensief onderzoek op deze lijn is dientengevolge opgenomen in dit onderzoek.

Op basis van de resultaten van hogervermelde proeven en enkele ongepubliceerde data op ons Instituut verzameld, werd besloten om een beperking tot 80 % voor 4 dagen startend vanaf dag 4 te gebruiken in alle volgende proeven. Na de periode van de beperking werden de dieren telkens terug *ad libitum* gevoederd.

Uit de resultaten van de proeven beschreven in hoofdstuk 6 kwam naar voren dat deze kwantitatieve voederbeperking inderdaad een goede methode is om voldoende compensatorische groei te induceren. In beide proeven was het eindgewicht van de beperkte Ross 208(308) dieren gelijkaardig of waren de dieren zelfs zwaarder dan de controlegroep. Bij de dieren van de 508-lijn werd een substantiële compensatorische groei gevonden in de eerst proef maar die bleef uit in de tweede proef. In tegenstelling met de resultaten van de vorige proeven kon de voederconversie in een aantal gevallen wel degelijk significant verbeterd worden. Echter wel in de lijn van vorige proeven was de trend tot verlaagde mortaliteit.

Bij compensatorische groei werd een verbeterde N-retentie gevonden. Ondanks het feit dat de verbeteringen statistisch niet significant waren, kunnen deze toch wel van belang zijn voor het milieu. Bovendien bleek de N-retentie gecorreleerd met de lijn en de leeftijd van de dieren.

Er is slechts weinig bekend betreffende de eiwitbehoeften voor en tijdens de periode van compensatorische groei. In hoofdstuk 7 werd daarom dieper ingegaan op het effect van het eiwitgehalte van de starter enerzijds en het eiwitgehalte van de groeier (tijdens de compensatorische groei) anderzijds. Uit literatuur is reeds gebleken dat een verlaagd eiwitgehalte in de starter geen aanleiding geeft tot verminderde zoötechnische prestaties. Het is echter niet duidelijk in hoeverre de mogelijkheid tot inhaalgroei na een voederbeperking gecorreleerd is met het eiwitgehalte van de starter. Daartoe werd een klassieke starter met 22 % RE naast een laageiwitstarter met 20 % RE uitgetest in hoofdstuk 7.

Om na te gaan of er een verhoogde eiwitbehoefte bestaat bij voederbeperkte dieren tijdens de inhaalgroei, werd binnen deze starterbehandelingen een normale groeier (20 % RE) of een hoogeiwitgroeier (22 % RE) uitgetest. Er werden terug twee lijnen opgezet en dezelfde kwantitatieve beperking werd toegepast.

Bij het verlagen van het eiwitgehalte van de starter werden geen significante verschuivingen in de groeiprestaties of inhaalgroeicapaciteit vastgesteld. Er was wel een trend van verhoging van de voederconversie. Het feit dat een kwantitatieve voederbeperking deze verslechterde VC kon voorkomen heeft waarschijnlijk te maken met de verbeterde N-retentie en verminderde eiwit turn-over bij vleeskippen tijdens de inhaalgroeifase. Een verhoogd eiwitgehalte in de groeier gaf aanleiding tot een verdere verbetering van de VC. Vooral de Ross 508 lijn, gevoederd met een laageiwitstarter, bleek een hogere behoefte voor eiwit in de groeifase te kennen.

De invloed van het eiwitgehalte op de karkassamenstelling was duidelijk verschillend naargelang de lijn. Voor de Ross 308 dieren bleek de kwantitatieve voederbeperking een grotere impact op de karkassamenstelling te hebben dan het eiwitgehalte van de groeier. De voederbeperking had een significant positief (dalend) effect op het totale vetgehalte. Het effect op het abdominaal vetgehalte en borstvleespercentage was echter beperkt positief. Bij de 508-lijn bleek vooral het voordeel van een verhoogd eiwitgehalte in de groeier na voederbeperking belangrijk. Het eiwitgehalte van de groeier had een positief effect op totaal karkaseiwitgehalte en slachtrendement.

Bovenvermelde resultaten bevestigen het feit dat de resultaten van een voederbeperking vrij variabel kunnen zijn. Dit kan gedeeltelijk verklaard worden door het feit dat heel wat factoren hun invloed hebben wanneer deze vorm van groeisturing wordt beschouwd. Soort, periode, intensiteit en duur van de restrictie of genetische factoren zoals lijn en geslacht zijn factoren die reeds beschreven werden in de literatuur en verder werden geïllustreerd in dit onderzoek. Bij het elimineren van deze factoren zijn de resultaten van een groeisturing toch nog vrij onvoorspelbaar. De beschreven parameters zijn met andere woorden onvoldoende om de vastgestelde variatie te verklaren. In de hoofdstukken 8 en 9 werd dan ook onderzoek gedaan naar enkele additionele factoren die verder kunnen bijdragen tot de verklaring van de gevonden variatie.

Uit de resultaten van bovenstaande proeven kwam het vermoeden dat “kuikenkwaliteit” een belangrijke invloed kan hebben op de capaciteit van de kuikens om compensatorische groei te realiseren. In hoofdstuk 8 werd daarom onderzoek gedaan naar de invloed van de leeftijd van de ouderdieren, als een mogelijke oorzaak voor verschillen in kuikenkwaliteit, op de inhaalgroeicapaciteit van hun nakomelingen.

Uit literatuur blijkt inderdaad dat er een correlatie bestaat tussen het eendagskuikengewicht en de groeicapaciteit. Er werd in het verleden echter nog geen onderzoek gedaan naar de invloed van eendagskuikengewicht en de inhaalgroeicapaciteit na voederbeperking. De vraag stelt zich met andere woorden of gegevens van eendagskuikens gerelateerd zijn met hun capaciteit om compensatorische groei te realiseren.

Drie verschillende leeftijden ouderdieren werden gebruikt in twee opeenvolgend proeven met Ross 308 en Ross 508 dieren. Kuikens van jonge (± 32 weken), gemiddelde leeftijd (± 45 weken) en oude (± 60 weken) ouderdieren werden in beide proeven opgezet. Deze verschillen in leeftijden gaven aanleiding tot significant verschillen in de kenmerken van de eendagskuikens. Er werd van uitgegaan dat dit verschillen in kuikenkwaliteit creëerde.

De invloed van de leeftijd van de ouderdieren op de compensatorische groei was zeer uiteenlopend tussen de twee opeenvolgende proeven. Kuikens van de jonge moederdieren realiseerden een uitgesproken inhaalgroei in de eerst proef. Dit kon echter niet bevestigd worden in de tweede proef. Bovendien toonden de resultaten dat waarschijnlijk een strengere beperking nodig is voor de kuikens afkomstig van de oudere moederdieren. Er kan besloten worden dat de leeftijd van de ouderdieren van de kuikens van invloed is op de compensatorische groeicapaciteit maar er rekening moet gehouden worden met eventuele interacties met de proefomstandigheden.

In hoofdstuk 9 werd de invloed van de voederstructuur bekeken als een mogelijk andere factor van invloed. Twee experimenten werden uitgevoerd met terug dezelfde Ross lijnen om na te gaan of er verschillen in compensatorische groei mogen verwacht worden bij gebruik van een korrel- of een meelvoeder bij een kwantitatieve voederbeperking. De leeftijd van de moederdieren in de eerste proef was respectievelijk 46 en 44 weken voor de Ross 308 en Ross 508 kuikens. In de tweede proef hadden beide groepen ouderdieren een leeftijd van 43 weken.

In beide proeven en voor beide lijnen werden geen significante effecten van de restrictie op eindgewicht of VC gevonden. Korrelvoedering daarentegen gaf aanleiding tot hogere eindgewichten en verbeterde VC in vergelijking met meelvoedering. Compensatorische groei bleef uit bij korrelgevoederde dieren. Bij gebruik van meelvoeders werd wel inhaalgroei vastgesteld hoewel niet in alle gevallen. Ook het tijdstip van aanvang van compensatorische groei was verschillend.

Ondanks de hogere groei bij voedering van korrels werd geen significante stijging van de sterfte vastgesteld. Voederbeperkingen daarentegen gaven echter terug aanleiding tot een trend van verlaagde sterfte. Er waren geen aanduidingen dat dit positieve effect verschillend was naargelang de voederstructuur.
Er werden geen eenduidige effecten van de voederbeperkingen op de karkassamenstelling gevonden. Het gebruik van korrels daarentegen had een positief effect op het slachtrendement en het borstvleespercentage. In de tweede proef was er echter wel een aanduiding van een verlaagd slachtrendement en borstvleespercentage bij combinatie van voederbeperking met een korrelvoedering.
Algemeen kan er besloten worden dat meerdere factoren een rol spelen in de capaciteit van de kuikens om compensatorische groei te realiseren na een vroege, tijdelijke voederbeperking. Bovendien wordt niet in alle gevallen een significante verbetering van de VC en een significante verlaging van het karkasvetgehalte gerealiseerd. De gevonden variatie kan gedeeltelijk verklaard worden door voederstructuur en “kuikenkwaliteit”. Toch blijkt het, in de gegeven omstandigheden, onmogelijk om de uiteindelijke resultaten te voorspellen. Met andere woorden, het is voor de pluimveehouder zeer moeilijk om te beslissen of er moet gekozen worden voor een voederbeperking of niet bij de aankomst van de kuikens. Er kan evenwel geadviseerd worden om de groei eerder continu te gaan sturen door de voederopname dagelijks te gaan aanpassen in plaats van een a priori vastgelegde voederbeperking toe te passen in alle omstandigheden.

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Curriculum vitae

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- lager secundair onderwijs : 1982-1985
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1 april 2002 – 30 september 2002	:	assistente, contractueel, Ministerie van Middenstand en Landbouw
1 oktober 2002 -	:	assistente, contractueel, Ministerie van de Vlaamse Gemeenschap

<u>Begeleiding buitenlandse onderzoeker</u>

Begeleiding van Garba Laouali (Niger) in het kader van een Master of Science thesis (Tropisch Instituut - Antwerpen) met als onderwerp : ‘Sturing van de groeicurve bij vleeskippen”. 1998-1999.

<u>Externe dienstverlening</u>

Medewerking aan een onderzoek naar de biobeschikbaarheid van niet-organische mineralenbronnen in de veevoeding in samenwerking met ID-TNO Animal Nutrition (Nederland) en INRA (Frankrijk) onder leiding van EMFEMA.

List of publications

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